Juvenile nasopharyngeal angiofibroma

Clinical profile and analysis of recurrence

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A dissertation submitted in part fulfillment of MS Branch IV, ENT examination of the Tamil Nadu Dr. MGR Medical University, to be held in March 2009

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Certificate

This is to certify that the dissertation entitled, 'Juvenile Nasopharyngeal Angiofibroma: Clinical profile and analysis of recurrence' is the bonafide original work of Dr. Sunil Jalan submitted in fulfillment of the rules and regulations for the MS Branch IV, ENT examination of the Tamil Nadu Dr. MGR Medical University, to be held in March 2009.

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And to God be the glory, great things He hath done.

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INTRODUCTION

Juvenile nasopharyngeal angiofibroma (JNA) is a fibrous and vascular tumour that commonly involves the nasopharynx.[1] It accounts for < 0.5% of head and neck tumours and primarily affects male adolescents.[2] It is a nonencapsulated, submucosal spreading tumour which is locally destructive. Originating from the superior posterior margin of the sphenopalatine foramen, progression of the tumour is often slow with extension into the pterygopalatine fossa, the paranasal sinus or infratemporal fossa.[3]

Recurrent severe epistaxis accompanied by progressive nasal obstruction is the classical symptom of juvenile angiofibromas at the time of presentation. Other signs and symptoms of tumour growth and extension may include swelling of the cheek, trismus, hearing loss, anosmia and a nasal intonation or plumy quality of the voice. More extensive tumour growth with invasion of orbit and cavernous sinus can cause proptosis, diplopia, visual loss, facial pain and headache.[4]

For tumour evaluation towards staging and planning for treatment, contrast computed tomography (CT) and magnetic resonance imaging (MRI) reliably assess tumour extent. Preoperative arteriography is helpful to evaluate feeding vessels and allows embolization of these highly vascular lesions.[5]

The mainstay treatment of JNA is surgery, although radiotherapy, hormone therapy, cryotherapy, electrocoagulation, and chemotherapy have all been described in the literature. Most authors prefer external surgical approaches and reserve radiation for

unresectable intracranial tumours or as post-operative adjunctive therapy when residual tumour has to be left behind.[6]

Intraoperative bleeding is one of the major concerns during operation. In an attempt to reduce intraoperative bleeding to a minimum, transcatheter embolization of JNA is often undertaken preoperatively.[7]

Recurrence is by far the most common complication encountered and may develop in up to 40 percent of these patients.[8]

This study was undertaken in a tertiary care hospital in South India, to describe the clinical profile of patients who were diagnosed to have juvenile nasopharyngeal angiofibroma and to assess the factors associated with its recurrence.

AIMS OF THE STUDY

1. To describe the clinical profile of patients diagnosed to juvenile nasopharyngeal angiofibroma.

2. To determine the rate of recurrence and to analyze factors involved in recurrence.

3. To analyze factors affecting intra-operative blood loss.

OBJECTIVES OF THE STUDY

(a) To describe the spectrum, frequency and duration of symptoms in patients diagnosed to have juvenile nasopharyngeal angiofibroma.

(b) To evaluate the extent of tumour involvement at presentation on imaging.

(c) To determine the relationship between staging of tumour and

i. Age

ii. Duration of symptoms prior to presentation

(d) To evaluate factors that predicts recurrence of disease.

(e) To describe the clinical profile of patients who present with recurrence of tumour after primary surgical treatment.

(f) To evaluate the extent of tumour involvement at the time of recurrence by imaging.

(g) To compare the clinical profile of primary and secondary (recurrent) cases.

PRESENT KNOWLEDGE AND REVIEW OF LITERATURE

Introduction and historical aspects

Juvenile nasopharyngeal angiofibroma (JNA) is a fibrous and vascular tumour that commonly involves the nasopharynx.[1] Juvenile nasopharyngeal angiofibroma (JNA) accounts for < 0.5% of head and neck tumours and primarily affects male adolescents.[1] It is a nonencapsulated, submucosal spreading tumour which is locally destructive. Originating within the superior posterior margin of the sphenopalatine foramen, progression of the tumour is often slow, uneventful, and undetected until the tumour has extended into the pterygopalatine fossa or the paranasal sinus, or with the occurrence of atypical symptoms.[1] Nasopharyngeal angiofibromas have been identified since ancient times and Hippocrates (470 – 471 BC) removed the "hard nasal polyp" through a midline, nasal-splitting incision. The first successful surgical treatment (total maxillectomy) is credited to Liston at the University College Hospital in London in 1841. The term "nasopharyngeal fibroma" was first coined by Chauveau.[9] Friedberg suggested the name 'angiofibroma' in 1940.[1]

Site of origin and spread of tumour

The point of tumour origin is important in that its location helps predicts tumour growth and influences the surgical approach and tumour excision. A number of theories have been propounded over the years to explain the origin of angiofibromas and although one or two are seemingly plausible, none is entirely convincing. Ringertz (1938) suggested that the tumour arouse from the periosteum of the nasopharyngeal vault, while Som and Neffson (1940) believed that inequalities in the growth of the bones forming the skull

base resulting in hypertrophy of the underlying periosteum in response to a hormonal influence. Bensch and Ewing (1941) thought that the tumour probably arose from embryonic fibrocartilage between the basiocciput and basisphenoid. Brunner (1942) believed an origin from the conjoined pharyngobasilar and buccopharyngeal fascia. Osborn (1959) considered two alternatives, namely the possibility that the swellings were either hamartomas, or residues of fetal erectile tissue which were subject to hormonal influences. Girgis and Fahmy (1973) noted cell nests of undifferentiated epithelioid cells or 'zellballen' at the growing edge of angiofibromas, an appearance which they likened to that of paragangliomas. They also commented on the existence of paragangliomatous tissue around the terminal part of the maxillary artery in the pterygopalatine fossa of stillborn infants.[10] The occurrence of these rare tumours almost exclusively in adolescent males supports the hypothesis that an alteration of the pituitary androgen-estrogen axis contributes to the pathogenesis of JNA.[11] Exhaustive studies of the pituitary- gonadal axis in these patients, however, have failed to identify any endocrinologic abnormality. The most widely accepted theory of origin of JNA is that the tumour is derived from embryologic chondrocartilage during the development of the cranial bones.[11] This tumour is said to originate form the superior margin of the sphenopalatine foramen and has a tendency to grow and extend along the natural foramina and fissures associated with its site of origin. From its origin, the tumour can grow unimpeded into the nasopharynx and nasal cavity, invade maxillary, ethmoid, or sphenoid sinuses by bone destruction, and spread laterally into the pterygopalatine fossa. From here, the tumour can erode the pterygoid plates leading into the infratemporal fossa or grow through the inferior orbital fissure into the orbit. From the infratemporal fossa, superior extension of tumour would

involve the pterygoid process and the foramina rotundum, ovale, and lacerum to involve the middle cranial fossa. From the middle cranial fossa, the tumour can invade the parasellar region, usually remaining extradural and lateral to sphenoid sinus. There are two patterns of intracranial extension. The first is through destruction of skull base at the attachment of pterygoid process, lateral to the internal carotid artery. The second is through the sphenoid sinus and into the region of the cavernous sinus.[12]

Pathogenesis

As this tumour is almost exclusively found in adolescent boys, there has always been much speculation and indirect evidence that sex-hormone receptors play some part in its development. Recent immunocytochemical techniques have been used to show that androgen receptors are present in at least 75 percent of tumours, these receptors being present in both vascular and stromal elements. A much smaller proportion of tumours also have some progesterone receptors. In contrast, oestrogen receptors have not been demonstrated. Other factors also play their part in the development of this tumour. The angiogenic growth factor (Vascular Endothelial Growth Factor (VEGF)) has been found localized on both endothelial and stromal cells, perhaps indicating that both cell types play a role in tumour development. Vessel density and both the expression and localization of VEGF correlate with the proliferative marker Ki67. However, neither the proliferative index nor VEGF expression seems to bear any relation whatsoever to the stage of tumour at time of presentation; in other words, its degree of aggressiveness. Over expression of insulin-like growth factor II (IGF II) has also been found in a large number

of juvenile angiofibromas. It is thought the over-expression of IGF II might be associated with a tendency to recurrence and poorer prognosis.[4]

Symptoms and signs

Recurrent severe epistaxis accompanied by progressive nasal obstruction is the classical symptoms of juvenile angiofibromas at the time of presentation. Epistaxis may vary in severity from the occasional show to an alarming and sometimes a life-threatening event. Chronic anaemia is thus a common feature of the established condition. Bleeding which occurs prior to surgery is entirely spontaneous and usually unconnected with trauma. Nasal obstruction is often complete such that stasis of secretions and sepsis are virtually inevitable, followed by hyposmia and anosmia. The voice acquires a nasal intonation and, if the swelling is large enough to force the soft palate down, there may be an added plummy quality to it. Blockage of the eustachian tube is not uncommon in such a situation and leads to deafness and Otalgia.[10]

Anterior rhinoscopy is likely to confirm the presence of abundant mucopurulent secretions together with bowing of the nasal septum to the uninvolved side. Posterior rhinoscopy in the cooperative relaxed patient usually displays a pink or red mass filling the nasopharynx, but the sheer bulk of the lesion rarely allows the examiner to determine its precise site of origin. Gross physical signs are evident when extensive disease has involved the nose and infratemporal fossa. The nasal bones are often splayed out and there may be obvious swelling in the temple and cheek. Intraoral palpation in the interval between the ascending ramus of the mandible and the side of maxilla may also reveal a

fullness caused by a tumour which has crept around the back of antrum. Impaction of bulky disease in the infratemporal fossa results in extreme signs such as trismus and bulging of the parotid gland, while proptosis is a definite sign that the orbital fissures have been penetrated. The classical frog face is the ultimate picture of massive escape of disease. Headache is not uncommon in patients with large tumours and is often attributable to chronic sinusitis. Falling vision indicates tenting of the optic nerve over a substantial extra nasopharyngeal extension of the tumour.[10]

These tumours do not grow fast and so many months or even years may pass before it occurs to the patient or their parents that there is anything seriously amiss. In most, there is a delay of at least six or seven months between the onset of symptoms and presentation.[4]

Histopathology

The gross appearance of the neoplasm is of a lobulated, pink to purplish mass with a smooth surface. They are covered by intact mucosa on their nasopharyngeal surface (unless previously biopsied). The cut surface has a fibrous appearance, often with blood vessels seen at the base of resection. Histologically, the tumour consists of two main cellular components, a fibrous stroma consisting of spindle or stellate-shaped cells in a dense collagen matrix, and a rich network of irregularly shaped blood vessels. The vessels vary from small endothelial-lined capillaries to large venous channels. A characteristic feature of these channels is a distinct lack of smooth muscle and elastic fibers found in normal blood vessels, which contributes to the tumour's hemorrhagic

propensity after even minimal manipulation. The stromal cells appear to be of fibroblastic and myofibroblastic origin.[13]

Diagnosis

During initial evaluation, contrast computed tomography (CT) and magnetic resonance imaging (MRI) reliably assess tumour extent. CT diagnosis is based on two constant features that include (1) mass in nose and pterygopalatine fossa and (2) erosion of bone behind the sphenopalatine foramen at the root of the pterygoid plate.[1] Areas inaccessible to clinical examination, such as posterior part of nasal cavity, the sphenoid and ethmoid sinuses, the pterygopalatine fossa, the middle cranial fossa and sometimes the nasopharynx, are delineated well on axial CT cuts. Of particular help is the ability of CT to reveal erosion of the sphenoidal sinus and the skull base - both potential routes of intracranial extension which may be a source of recurrence if left unrecognized. CT demonstrates well widening of the pterygopalatine fossa - a sign strongly suggestive of JNA. With expansion of the tumour, anterior bowing of the posterior antral wall may be seen on plain radiographs and CT. This is the ‘antral sign’ described by Holman and Miller in 1965. The tumour’s attenuation coefficient of 35 Hounsefield numbers enables orbital and intracranial involvement to be easily detected since, in both these areas, the surrounding tissues have smaller attenuation coefficients. Hence preoperative CT scanning is a vital adjunct in deciding on the approach, particularly in recurrent cases where previous surgical attempts and scarring invariably alter the true picture.[14]

Magnetic resonance imaging with gadolinium enhancement allows the surgeon to more accurately stage the lesion and plan for surgical approach. It offers additional information

about possible intracranial and cavernous sinus extension.[11] It provides multiplanar imaging; improved definition at the cribriform plate and cavernous sinus, superior differentiation of the tumour from inflamed mucosa and fluid in sinuses, and avoidance of diagnostic radiation in patients who require serial follow up.[15] The main advantage of MRI, however, lies in post-treatment surveillance of residual tumours or recurrent disease.[13] Preoperative arteriography is helpful for the evaluation of feeding vessels and allows embolization of these highly vascular lesions.[5]

Biopsy is rarely required to establish the diagnosis and should be avoided because of the risk of hemorrhage.[15] Despite classic radiographic findings, there is no absolute radiological sign of nasopharyngeal angiofibroma. If the tumour appears atypical or if patient's clinical history is unusual, then biopsy should be considered before tumour resection. This should be performed in the operation theatre as significant hemorrhage may require anterior and post nasal packing or cauterization and is best handled in a controlled environment. Fibrous dysplasia, lymphoepithelioma, and rhabdomyosarcoma have been known to mimic nasopharyngeal angiofibroma.[11]

Staging system for JNA

While there is usually a linear relationship between tumour size and extranasopharyngeal extension, size is not consistently measurable. As in malignant neoplasms of the nasopharynx, tumour staging is based on sites of involvement. Staging is important for individual evaluation as well as inter institutional treatment comparisons. Several staging system are proposed.

Chandler et al (1984)[16]

Stage I	Tumour confined to the nasopharyngeal vault
Stage II	Tumour extending into nasal cavity and/or sphenoid sinus
Stage III	Tumour extending into antrum, ethmoid sinus, pterygomaxillary fossa, infratemporal fossa, orbit and or cheek
Stage IV	Intracranial tumour

Sessions et al (1981)[17]

Stage I A	Tumour limited to posterior nares/or nasopharyngeal vault
Stage I B	Tumour involving posterior nares and/or nasopharyngeal vault with involvement of at least 1 paranasal sinus
Stage II A	Minimal lateral extension into pterygomaxillary fossa
Stage II B	Full occupation of pterygomaxillary fossa with or without superior erosion of orbital bones
Stage II C	Infratemporal fossa with or without cheek
Stage III	Intracranial extension

Radkowski (1996)[11]

Stage I A	Tumour limited to nose and/or nasopharyngeal vault
Stage I B	Tumour involving nose and/or nasopharyngeal vault with involvement of at least 1 paranasal sinus
Stage II A	Minimal lateral extension into pterygomaxillary fossa
Stage II B	Full occupation of pterygomaxillary fossa with or without superior erosion of orbital bones
Stage II C	Infratemporal fossa or posterior to pterygoid plates
Stage III A	Erosion of base of skull (middle cranial fossa/base of pterygoids) – minimal intracranial extension
Stage III B	Extensive intracranial extension with or without extension into the cavernous sinus

Classification according to Fisch[10]

Stage I	Tumours limited to nasal cavity, nasopharynx with no bony destruction
Stage II	Tumours invading pterygomaxillary fossa, paranasal sinuses with bony destruction
Stage III	Tumours invading infratemporal fossa, orbit and/or parasellar region remaining lateral to cavernous sinus
Stage IV	Tumours invading cavernous sinus, optic chiasmal region, and/or pituitary fossa

Andrews staging of angiofibroma[18]

Stage I	Tumour limited to the nasal cavity and nasopharynx
Stage II	Extensions into pterygopalatine fossa, maxillary, sphenoid, ethmoid sinuses
Stage IIIa	Extensions into orbit or infratemporal fossa without intracranial extensions
Stage IIIb	Stage IIIa with small extradural intracranial (parasellar) involvement
Stage IVa	Large extradural intracranial or intradural extensions
Stage IVb	Involvement of the cavernous sinus, pituitary, or optic chiasm

Chandler's staging system was based on a system proposed for nasopharyngeal cancer. JNA, however, is a benign lesion with a predictable growth pattern that differentiates it from malignant nasopharyngeal tumours. In Chandler's staging system stage III includes all extranasopharyngeal sites except sphenoid sinus and intracranial extension. The inclusion of multiple sites with variable resectability into a single stage limits the clinical usefulness of the system.[11]

Sessions system of classification more accurately reflects the clinical behavior of JNA.[17] In 1993, Radkowski further modified Sessions classification to include posterior extension to pterygoid plates and the extent of skull base erosion.[11]

Treatment

The mainstay treatment of JNA is surgery, although radiotherapy, hormone therapy, cryotherapy, electrocoagulation, and chemotherapy have all been described in the literature. Most authors prefer external surgical approaches and reserve radiation for unresectable intracranial tumours or as post-operative adjunctive therapy when residual tumour has to be left behind.[6]

Until relatively recently, most small tumours were resected either through a transpalatal approach, lateral rhinotomy or mid-facial degloving approach. Open approaches can be used for tumours of all stages and certainly were only option before the application of endonasal endoscopic techniques became more widespread.[19]

The treatment should take into account the following factors: 1) tumour growth in prepubescent boys may give rise to significant morbidity, 2) a wide approach to the skull base may also result in significant morbidity, and 3) JNA has an unusual natural history in which tumour remnants may become involuted or stabilize with maturity.[19]

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Surgical approaches for JNA

Various surgical approaches are described for excision of JNA including endoscopic transnasal, transpalatal, medial maxillectomy, facial translocation and infratemporal fossa approaches with or without craniotomy.[15] From a practical standpoint, the choice of surgical approach is based heavily on the surgeon's experience and training. However, several basic concepts are critical: adequate exposure of the tumour, ability to control

bleeding, no postoperative facial scar or deformity, and no interference with the growth of the facial skeleton. Exposure of the tumour and hemostasis are a high priority and often are cited as the justification for aggressive and potentially deforming transfacial approaches. Avoidance of scars, deformity, or interference with facial growth has gained greater importance as treatment philosophies that emphasize quality of life have evolved, with surgeons developing techniques that minimize functional as well as cosmetic deficits.[20]

Transpalatal

The transpalatal approach was formerly used for removal of nasopharyngeal angiofibromas, but it is not as popular as it once was.[21] It is suited for tumour limited to the nasopharynx, nasal cavity and sphenoid sinus because lateral exposure is limited.[13]

The best incision is one that is placed just inside the upper alveolus to provide a large palatal flap. The incision is made down to the bone with a knife and the soft tissue is elevated from the bone backwards to the posterior edge of the hard palate. The exposure is increased by extending the incision backwards into the soft palate and if necessary dividing the greater palatine vessel on one side. The bony hard palate is then removed as necessary, using a cutting burr and bone-nibbler forceps. It is important that there is always bone underneath the incision line or else an oronasal fistula occurs. Since removal of bony hard palate is always necessary in this approach, it is important to make the incision as far forward as possible. A palatal fistula may results in as many as 40% of patients.[21]

Lateral Rhinotomy

Lateral rhinotomy is the traditional and standard technique for tumour of nose and paranasal sinuses. The lateral rhinotomy incision is effective for the exposure of the nasopharynx, paranasal sinuses, and pterygopalatine fossa and the medial parts of the infratemporal fossa, as well as cavernous sinus. This external approach can allow optimal access with extensive tumours, yet with significant detriments because it may interfere with facial development or impose various dysfunctions.[22]

The principle disadvantage of the approach is that it leaves a long facial scar. With careful incision and suturing, however the scar can largely be hidden in the natural creases and shadow area of the face.[23]

Midfacial degloving

Midfacial degloving operation was first published by Casson et al. and described by Howard and Lund. This approach provides good exposure to target area and excellent cosmoses by avoiding external incision.[23] The exposure of the lower part of midface including the nose and maxillary sinus up to the orbit is excellent. The limitation of this approach is that it affords relatively poor access to orbit, the lateral aspect of maxilla, and the ethmoid sinus. Complication of this approach includes hematoma under the cheek flap and nasal vestibule stenosis.[19] A study done by El-Banhawy el al, evaluated 15 patients with type III (Fisch classification) primary JNA treated with endoscopic assisted midfacial degloving approach. Thirteen patients had complete tumour clearance with no

esidual or recurrence during the follow-up period. Recurrence was detected in 2 patients at 3 and 8 months post-operatively.[6]

Extended osteoplastic maxillotomy

The osteoplastic maxillotomy is a versatile new approach that can provide extensive exposure to the central skull base and infratemporal fossa and corresponding intracranial anatomy. Briefly, the maxillofacial skeleton is partially exposed via a Weber-Fergusson incision. Osteotomies in the maxilla and zygoma completely disengage the maxilla from the facial skeleton. The maxilla is mobilized on the skin and soft tissue of the ipsilateral cheek, maintaining its vascularity. Medial positioning of the anterior osteotomy through the face of the maxilla determines the extent of exposure to the nasopharynx. The lateral osteotomy at the malar eminence determines the extent of exposure to the infratemporal fossa. Concurrent use of temporal craniotomy provides corresponding access to the cranial cavity. Hearing and facial nerve are preserved. Form and function of maxilla are restored using miniplate fixation.[24]

Infratemporal fossa approach

The Fisch infratemporal fossa type C approach offers excellent skull base exposure for extradural tumours. With this procedure, a postauricular approach is used to perform a mastoidectomy and subtotal petrosectomy. The mandibular branch of the trigeminal nerve is sectioned, and after tumour removal the middle ear structures are removed, middle ear obliterated and Eustachian tube closed. This approach allows the patient to avoid facial incisions and a craniotomy; but it results in a permanent ipsilateral

conductive hearing loss and chin numbness.[25] Benefits of this approach includes: (1) direct visualization of the internal carotid artery while removing tumour; (2) a very short working distance; (3) ideal illumination, magnification, and binocular vision with use of the operating microscope; and (4) the potential exposure of the cavernous sinus through limited extradural temporal lobe elevation.[26]

When the angiofibroma is limited to either the infratemporal or pterygopalatine fossa and is not encroaching upon the internal carotid artery, the infratemporal fossa type D1 and D2 approaches may be used. As they avoid dissection through the middle ear, conductive hearing is preserved. These approaches are used to remove tumours involving the anterior cranial fossa and orbital apex and are typically carried out through either an extended preauricular (D1) or hemicoronal (D2) skin incision. The principle benefits of this approach include; (1) direct tumour access within the fossae; (2) no conductive hearing deficit: (3) avoidance of visible facial scars: (4) maintenance of normal facial contour: and (5) the potential to be converted to type C approach if necessary. The deficits associated with the type D approaches are very limited.[26]

Endoscopic

Endoscopic surgery in the nasal cavity and paranasal sinuses initially was used to treat non-neoplastic disease (polyposis, acute and chronic sinusitis, etc). As the skills of endoscopic surgeons improved, these techniques were adopted to excise benign neoplasms and more recently to remove locally confined malignancies. Tumours that involve the ethmoid, maxillary or sphenoid sinus, the sphenopalatine foramen,

nasopharynx, pterygomaxillary fossa and have limited infratemporal fossa involvement are amenable to endoscopic resection. Angiofibroma that involve the orbit or middle cranial fossa are not ideal for endoscopic excision and require more extensive surgery involving an intracranial - extracranial approach.[5] New advancements with endoscopic techniques today permit a minimally invasive resection of the entire tumour mass with minimal blood loss or morphological disturbance. Endoscopic procedures combined with computer-assisted systems avert complications resulting from lengthy radiation treatments as well.[22] A study in 2005[5] revealed relatively less blood loss with endoscopic approach compared to traditional lateral rhinotomy approach. The relatively low level of blood loss may be due to meticulous nature of dissection. Even small amount of bleeding limit the surgeon's endoscopic view and thus careful attention to hemostasis is essential for a successful outcome. In addition, much of the blood loss in traditional approaches results from the incisions and osteotomies of surgical access. Gilles Roger et al[27] evaluated 20 patients operated exclusively with endoscopic surgical approach. Endoscopic surgery was associated with a shorter duration of hospitalization with most patients discharged within 48 hours after surgery. In addition, endoscopic surgery avoids the complications specifically related to different approaches with open surgery (Epiphora, dysesthesia, trismus, pain, and possible effects on facial growth).

Lefort I osteotomy

This technique involves creating a transverse facial osteotomy along the lines of the classic Le Fort I fracture and inferiorly displacing the palate to expose nasopharynx, clivus, and posteriorly the sphenoid sinus. Among the advantageous of this option over

the other approaches to this area are the ease, rapidity, and safety with which it can be performed and its excellent cosmetic and functional results.[23] Occasionally, there can be troublesome bleeding from a torn maxillary artery which may be difficult to deal with via this approach. While it gives adequate access, it may need intramaxillary fixation postoperatively and the osteotomy is contraindicated under the age of 12 years because of the risk of the canine dentition. This degree of tissue damage is not really acceptable for angiofibroma.[21]

Role of radiation in JNA

Although radiation has been reported as an effective means of therapy for JNA, potential long term complications have dissuaded some from pursuing this modality of treatment. Secondary malignancies of the head and neck signify one of the most feared adverse sequelae of radiation exposure. Multiple papers have also reported deleterious effects on the visual system. However, it should be recognized that in many of these cases orbital involvement might have prevented proper eye shielding. The greater precision achieved with the recent development of three-dimensional conformal planning and intensity-modulation radiation therapy should succeed in more effectively shielding the lens from the irradiated field, even in context of significant orbital involvement.[28]

There has also been concern that radiation may affect growth centers in the face leading to abnormal development of the craniofacial skeleton, especially in the maturing adolescent. In view of the potential long-term complications of radiation therapy, many institutions still consider surgery to be the treatment of choice even for patients with

intracranial extension. Both extracranial and combined neurosurgical/otolaryngologic techniques have been described in the literature with varying degree of success. However, irrespective of whether extracranial or intracranial approaches are used, serious complications have been reported to ensue from surgical intervention. Life threatening hemorrhage, optic neurovascular bundle injury, cranial nerve damage, meningitis, and motor nerve deficits have all been reported.[28]

Preoperative chemotherapy

Oestrogens have been reported to induce shrinkage in some cases but their effect is variable and not without complication. At the very first, oestrogen therapy delays surgery and the secondary feminizing effects are certainly unwanted by an adolescent boy. In a small series of patients given the nonsteroidal androgen receptor blocker, flutamide, tumour shrinkage of up to 44 percent was reported by Gates et al.[29] Side effects of flutamide include nausea, breast tenderness and gynaecomastia. These effects were temporary and disappeared completely at the end of therapy. It appears that this drug might have a role in the preoperative preparation of patients with very advanced tumours, certainly those with intracranial extension. Unfortunately, in a pilot study of seven patients with stage IV disease the mean shrinkage achieved was 7.5% and this was considered to be insignificant.[4]

Embolization

As JNA is highly vascular, intraoperative bleeding is one of the major concerns during surgery. In an attempt to reduce intraoperative bleeding to a minimum, transcatheter

embolization of JNA is often undertaken preoperatively. The vascular supply of the JNA depends on the size and extension of the tumour. In the initial stages, when the tumour grows in the anterior nasopharynx and posterior portion of nasal cavity, there is a constant blood supply from the distal internal maxillary artery and its branches extending to the nasopharynx and nasal cavity (sphenopalatine and pterygopalatine arteries). As the tumour grows and involves more regions (sphenoid sinus, parapharyngeal spaces, etc.), other vessels, from both the internal and external carotid arteries, contribute to its vascular supply.[2] The goal of preoperative endovascular embolization is to achieve tumour devascularization whilst preserving a normal vascular supply to the surrounding tissues. This may be accomplished by the selective obliteration of the intratumoural vascular net. Reduction in peroperative blood loss after embolization may facilitate exposure of the tumour and the anatomical identification of the important structures during surgery, increasing the chances of achieving a radical tumour removal.[2] Severe complications like cerebral infarct and vision loss have been reported in the literature after tumour embolization with various techniques, which are generally secondary to dangerous collaterals from the internal maxillary artery to the intracranial/ intraorbital contents.[30]

Recurrence

Recurrence is commonly encountered following surgery and may develop in up to 40 percent of these patients. Not surprisingly, recurrence is more likely in patients with advanced disease and in those treated by inexperienced surgeons.[4]

Factor predicting rate of recurrence

(1) Age of presentation: Petruson et al[2] described that patients who were young at diagnosis were more likely to have recurrence than a patient who was older when the tumour was diagnosed.

(2) Anatomical factors: On the basis of review of preoperative CT and MRI, Lloyed et al.[31] determined that 93% of recurrence occurred in patients with invasion of sphenoid dipole through pterygoid canal. A further study in 2001[32] evaluated 19 patients undergoing surgical resection of angiofibroma and meticulous exploration and drilling of basisphenoid bone. Follow up of this cohort revealed no recurrence in this group, provoking the conclusion that careful attention to basisphenoid and pterygoid canal could nearly eliminate all residual disease. Deeper extension along the cancellous bone of the sphenoid and the pterygoid palates increases the likelihood that tumour remnant may be left behind at the time of surgery. Careful attention to this area and drilling of the bone surrounding the pterygoid canal intraoperatively may reduce the chance of leaving a tumour remnant in this area. Recurrence is inextricably linked to intracranial extension. Most studies cite a 10-20% incidence of intracranial extension, with a rate of nearly 50% of recurrence in these patients. On the basis of high rate of recurrence with intracranial extension, a combined craniofacial approach in needed in these patients.[5]

(3) Embolization: Some authors found that preoperative embolization reduced recurrences as well as intraoperative blood loss at primary surgery. McCombe et al. found that the strongest predictor of recurrence to be preoperative embolization, as embolization shrinks the tumour but makes complete excision more difficult, especially if there is deep invasion of the sphenoid.[2]

Treatment of recurrence

The presence of persistent radiographic abnormalities does not necessary signify residual tumour. Only if the patient develops symptoms or if new radiographic abnormalities arise is it necessary to pursue further treatment.[28]

Hemorrhagic risk

The resection of angiofibroma is classically considered to be high risk surgery particularly with respect to hemorrhage that may require blood transfusion, which is not without morbidity. In addition, perioperative bleeding can significantly hinder the excision.

The greater availability of procedures for autologous blood donation has allowed the use of heterogenous transfusions to be avoided. However, collecting autologous units can delay surgery by several weeks. Further this may be hindered by significant epistaxis. The risk of perioperative bleeding is greater at certain sites of dissection, for example, adjacent to the internal maxillary artery, the body of sphenoid sinus, and the roots of the pterygoids, the interpterygoid fossa as well as in the region of cavernous sinus.[4]

Complications

Postoperative complications varied and depended on the extent of surgery performed. A palatal fistula may result when the transpalatal route is used, especially if the incision is sited directly over the junction of hard and soft palate. Surgically induced infraorbital nerve sensory deficits are recognized as a potential complication of mid-facial degloving, as is nasal vestibular stenosis. Prolong nasal crusting is also common and this may well

develop into ozaena. With more extensive resections, ocular problems may be experienced including displacement of the globe caused by loss of bony support, ophthalmoplegia and visual loss.[4] Other reported complications are postoperative hemorrhage, meningitis, cerebrospinal fluid leakage, dental malocclusion, rhinolalia aparta, lacrimal duct stenosis and secretory otitis media.[7]

MATERIALS AND METHODS

Study Design: Non-concurrent prospective cohort study

Study population: All patients with histological proven juvenile nasopharyngeal angiofibroma, who underwent either primary or secondary surgical treatment at our institution (Christian Medical College, Vellore) between 1998 and 2008

Inclusion criteria: All patients with histologically proven JNA who underwent surgical treatment at our hospital over the specified period

Exclusion criteria: Patients with a diagnosis of JNA who did not undergo surgical treatment.

Informed Consent: Informed consent was obtained from all patients enrolled in the study. The consent form is attached as Appendix A. Institutional Research Board (IRB) approval was obtained from the institution for the conduct of the study.

Operational Definitions: For the purposes of this study,

1. Primary cases were defined as patients who were newly diagnosed to have juvenile nasopharyngeal angiofibroma and did not have any previous surgical intervention prior to presenting to our institution.

2. Patients with recurrence were defined as those who already had primary surgical treatment either in this institution or elsewhere and who manifested clinical symptoms or signs of recurrence.

Methodology

Step 1: Identification of patients

a) A search of the database of hospital admissions with a diagnosis of JNA was performed by medical records to identify index cases (both primary and secondary cases as defined above)

b) In addition a systematic search of the operation records of the ENT department during the period of interest (1998-2008) was done to identify any missed cases.

c) New cases of JNA that were diagnosed in the department during the study period (2007-2008) were also included

Step 2: Patient contact to collect and update data

a) New patients recruited during 2007-2008 were approached either in the outpatient clinic or in the ward for willingness to participate in the study

b) Patients who were already operated and who presented for follow up during 2007-2008 were approached for willingness to be part of the study

c) Other patients were contacted by mail and/or by telephone to request for either a review at the hospital if possible or to answer certain queries by phone or mail regarding symptoms of recurrence. Consent was obtained during this process.

Step 3: Data collection and abstraction

a) Data was obtained from medical records and by patient evaluation during hospital visit
b) Data was entered in the proforma (attached)
c) Data included demographics, symptomatology, extent of tumour (determined by imaging), surgical approach, complications and symptoms of recurrence (if present)

Step 4: Statistical aspects

a) Sample size calculation and rationale

Sample size for this descriptive study was calculated using the formula

$$N = 4\ PQ\ (Za + Zb)\ 2 / d^2$$

Where

N is number of sample size

P is prevalence of recurrence of disease - 27 % (Herman et al_{19})

Q is complement of prevalence = 73

d is effective measure (difference between estimate and variation allowed) - 20%

Calculated sample size N = 77 for this study.

b) Analysis

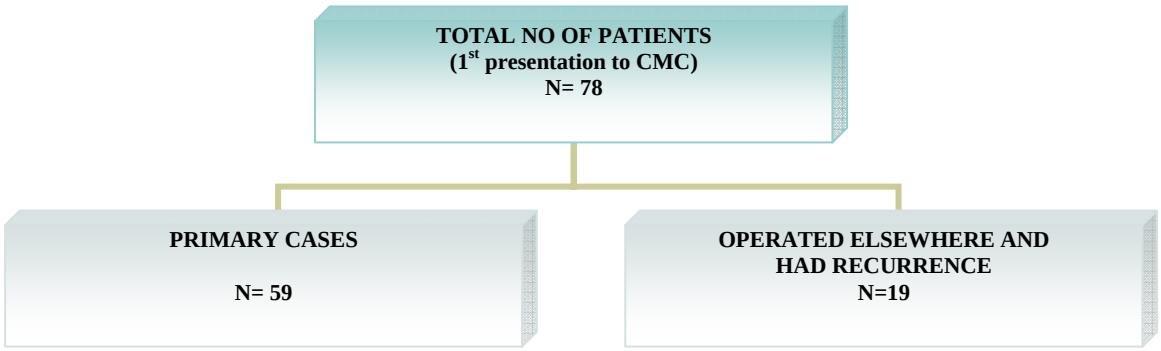
Statistical analysis was based on the appropriate statistical tests of significance. The χ^2 test and the Fisher's exact test were used for categorized bivariate analysis. The Student's t-test was used to compare the mean differences of the continuous variables. A P value of < 0.05 was considered as significant.

RESULTS

This study was conducted over a period of 19-months (April 2007 to October 2008) in the ENT Department of the Christian Medical College & Hospital (CMCH), Vellore. Patients with histologically proven Juvenile Nasopharyngeal Angiofibroma (JNA), who underwent surgical treatment in our institution from 1998, were identified from the hospital records. These patients were contacted by post and/or telephone and requested to present to hospital for review. In addition, new patients diagnosed to have JNA, who underwent surgical treatment in our hospital during the mentioned study period and who consented to participate in the study were also recruited.

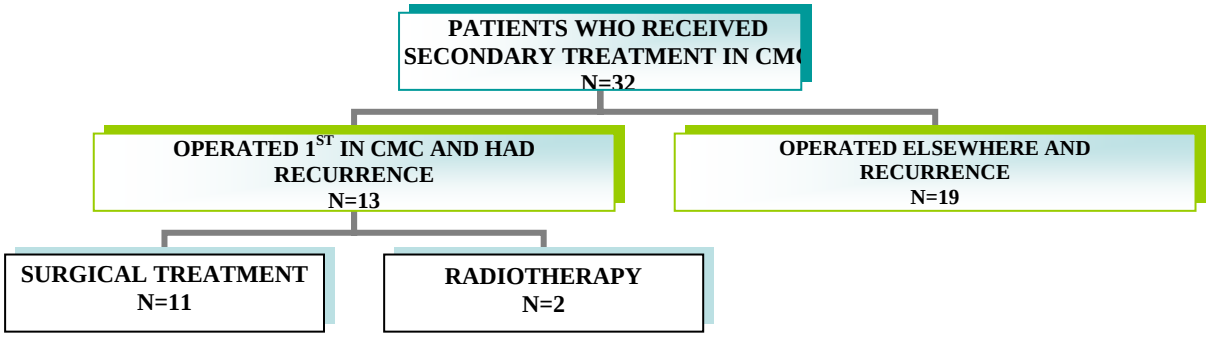
A total of 63 patients who had undergone surgical treatment at CMCH were identified from the hospital records. An additional 15 patients were recruited during the study period. Thus, a total of 78 patients with histologically proven juvenile nasopharyngeal angiofibroma who underwent surgical treatment at our institution formed the study cohort. Of these, 59 patients were newly diagnosed patients who had no surgical intervention prior to presentation at our institution. The remaining 19 patients presented with recurrence having had surgical treatment elsewhere (Figure 1). The mean (SD) duration of follow up of the primary cohort was 16.95 (20.32) months. The mean (SD) duration of follow up of patients with secondary disease was 18.77 (23.79) months.

Figure 1: Distribution of patients in the study



Thirty-two patients presented for secondary treatment following recurrence. This included 19 patients who came after treatment elsewhere and 13 patients who underwent surgical treatment in our institution for primary disease and subsequently had recurrence (Figure 2). Of these, 30 patients who underwent surgical treatment for recurrence of disease at our institution formed the secondary cases cohort, whist the two remaining patients who were treated with radiotherapy were excluded.

Figure 2: Distribution of patients who received secondary treatment in CMC



PRIMARY CASES

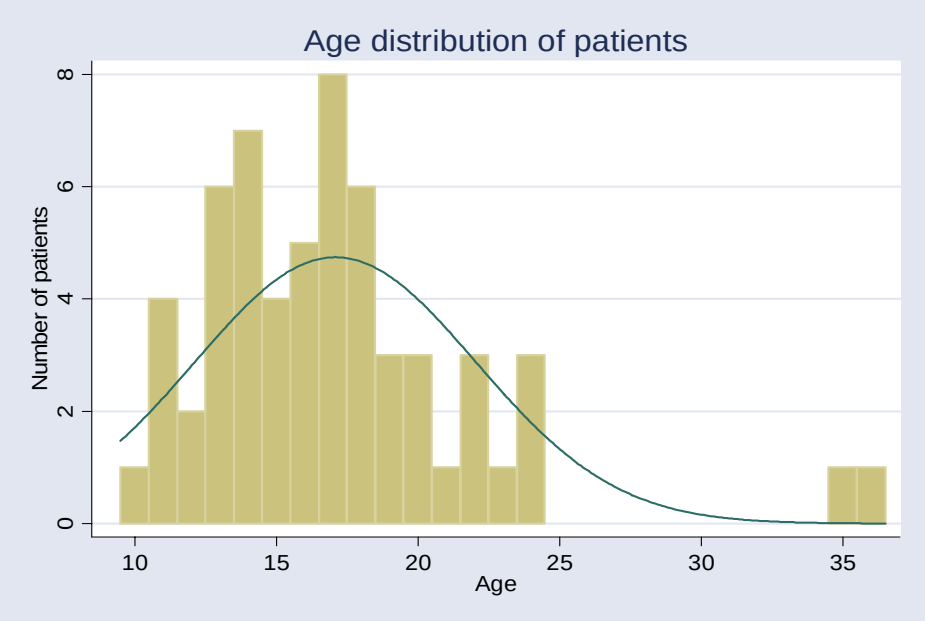
Demographics

The primary cases comprised of 59 patients with a mean (SD) age of 17.08 (4.96) years and a range of 10 to 36 years (Figure 3). The majority (83.1%) were of the age group between 10-20 years. There were 8 patients from 21- 30 years and 2 were above 30 years (Table 1). All patients were male.

Table 1: Age distribution of patients

Age group	Number of patients	Percentage
10-15 years	24	40.7
16-20 years	25	42.4
21-25 years	8	13.6
26-30 years	0	0
> 30 years	2	3.4
Total	**59**	**100**

Figure 3: Age distribution of patients (primary cases of JNA)



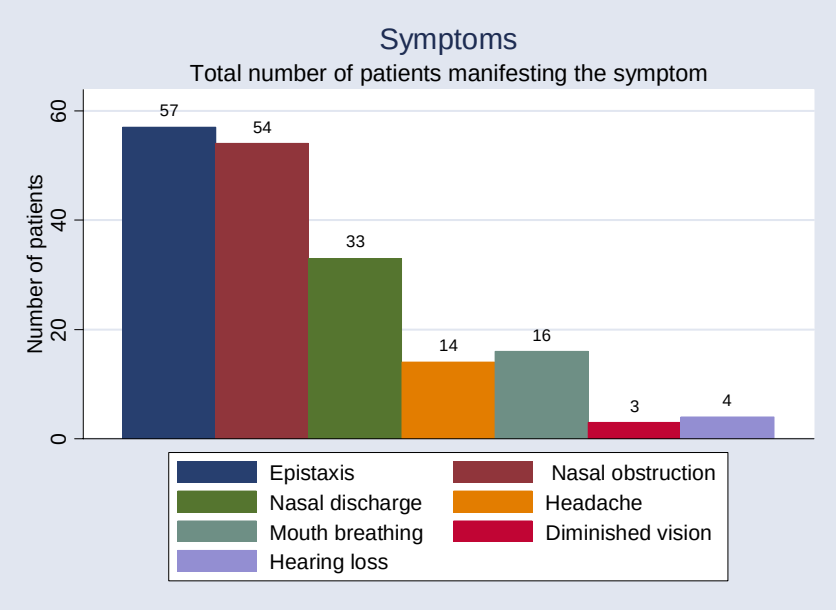
Symptomatology

The commonest symptoms that the patients presented (Table 2) with were epistaxis (96.6%) and nasal obstruction (91.5%). Other symptoms included nasal discharge, headache, mouth breathing, diminished vision and hearing loss (Figure 4).

Table 2: Spectrum of symptoms

Symptom	Number of patients	Percentage
Epistaxis	57	96.6
Nasal obstruction	54	91.5
Nasal discharge	33	55.9
Headache	14	23.7
Mouth breathing	16	27.1
Diminished vision	3	5.1
Hearing loss	4	6.8

Figure 4: Symptomatology



The mean duration of symptoms prior to diagnosis was about 7-8 months for epistaxis, nasal obstruction, nasal discharge, headache and mouth breathing (Figure 5). The symptoms that signified advanced disease like diminished vision and hearing loss had a shorter duration of symptoms prior to presentation (Table 3).

Figure 5: Duration of symptoms

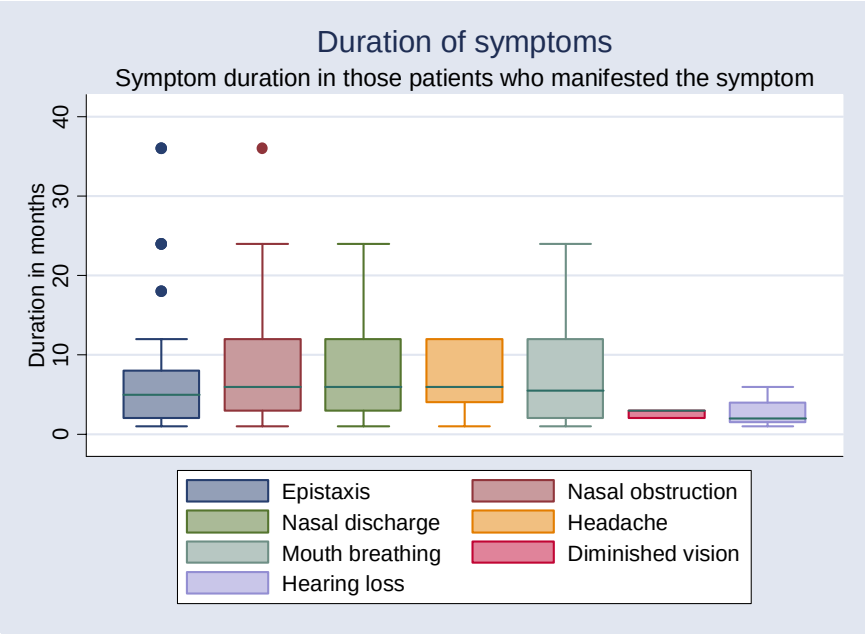
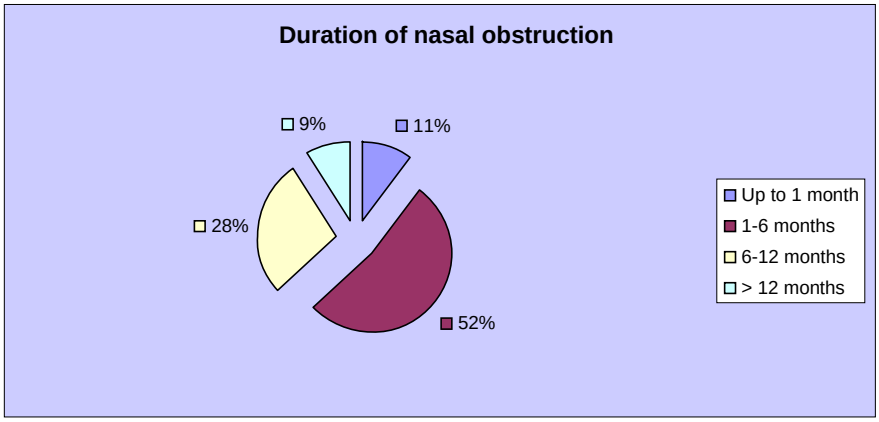


Table 3: Duration of symptoms

Symptom	No of patients (%)	Mean duration of symptom (months)	Standard deviation	Range (months)
Epistaxis	57 (96.6)	7.42	8.05	1 – 36
Nasal obstruction	54 (91.5)	7.83	7.11	1 – 36
Nasal discharge	33 (55.9)	6.79	4.87	1 – 24
Headache	14 (23.7)	6.79	3.95	1 – 12
Mouth breathing	16 (27.1)	8.12	7.63	1 – 24
Diminished vision	3 (5.1)	2.67	0.58	2 – 3
Hearing loss	4 (6.8)	2.75	2.22	1 – 6

Over 60% of patients who presented with epistaxis or nasal obstruction (Table 4) sought medical treatment within 6 months of onset of symptoms (Figure 6). Nearly 20% of patients with epistaxis came for treatment within one month of onset of symptoms. However 12.3% of patients with epistaxis and 9.3% of patients with nasal obstruction had symptoms for more than 12 months prior to treatment and presented late.

Figure 6: Duration of epistaxis and nasal obstruction



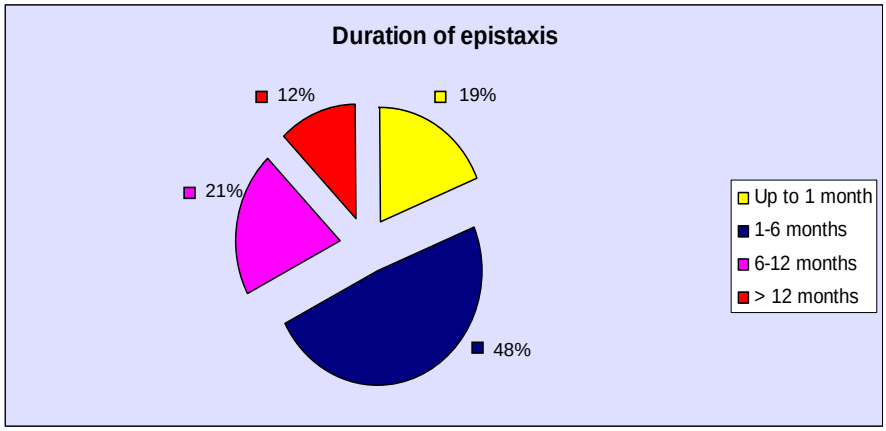


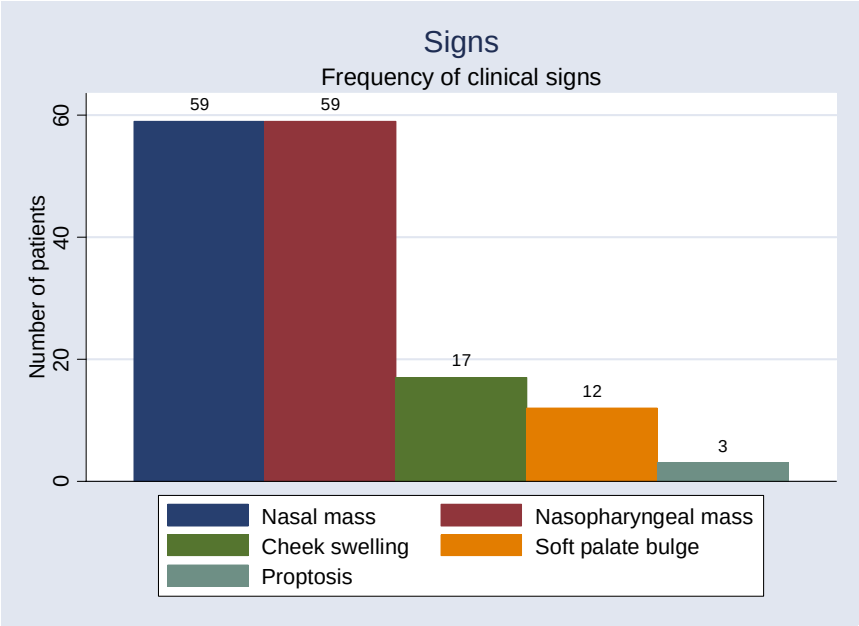
Table 4: Analysis of duration of epistaxis and nasal obstruction

Duration of symptom	No (%) of patients with epistaxis	No (%) of patients with nasal obstruction
Up to 1 month	11 (19.3)	4 (7.4)
1 to 6 months	27 (47.4)	29 (53.7)
6 to 12 months	12 (21.0)	16 (29.6)
> 12 months	7 (12.3)	5 (9.3)

Clinical signs

All patients had a nasal as well as nasopharyngeal mass (Figure 7). Other signs included cheek swelling (28.8%), soft palate bulge (20.3%) and proptosis (5.1%).

Figure 7: Frequency of clinical signs



Extent of tumour

Pre-operative **computed tomography** (CT) scans were done in 54 patients. In 5 patients, pre-operative scans were not done in view of clinically limited disease with endoscopic assessment. Of these 5 patients, 4 patients were found to have Stage I disease and 1 patient Stage II disease intra-operatively.

The nasal cavity and nasopharynx were found to be involved in all patients. Sphenoid sinus involvement was seen in 88% whilst the pterygopalantine fossa was involved in 66% of patients. The anatomic locations in which the tumour had extended are summarized in Table 5. Extradural involvement was seen in 10 patients (16.9%) and 8 patients (13.6%) had involvement of the cavernous sinus. The optic canal, foramen lacerum and pituitary fossa were not involved in any patient nor were intra-dural extension observed.

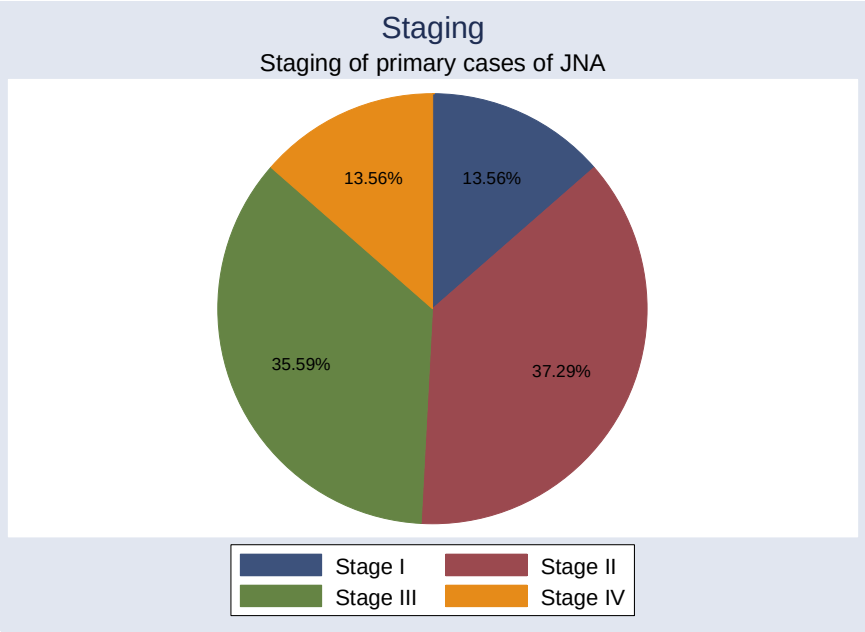
Table 5: Involved anatomic location of angiofibromas

Location *	Number	Percentage
Nasal cavity	59	100
Nasopharynx	59	100
Sphenopalantine region	53	88.1
Sphenoid sinus	39	66.1
Pterygoid palatine fossa	36	61.0
Infratemporal fossa	29	49.2
Ethmoid sinus	27	45.8
Base of pterygoid	21	35.6
Inferior orbital fissure	13	22.0
Pterygoid plate	10	16.9
Orbital apex	10	16.9
Extradural extension	10	16.9
Maxillary sinus	9	15.3
Foramen rotundum	9	15.3
Cavernous sinus	8	13.6
Interpterygoid fossa	7	11.9
Parapharyngeal space	6	10.2
Superior orbital fissure	5	8.5
Foramen ovale	3	5.1
Anterior cranial fossa	2	3.4
Clivus	1	1.7

* None of the patients had involvement of the optic canal, foramen lacerum, pituitary fossa or intradural extension

Fisch staging was used for the classification of JNA in this study. A small proportion of patients had early Stage I disease (13.6%). The majority of the cases operated were Stage II (37.3%) and Stage III (35.6%) disease and 13.6% of the patients had Stage IV tumour (Figure 8).

Figure 8: Stage of the disease



In this study, we observed that there was a trend (P=0.065) towards younger patients presenting with more advanced disease (Figure 9, Table 6).

Figure 9: Correlation between stage of disease and age

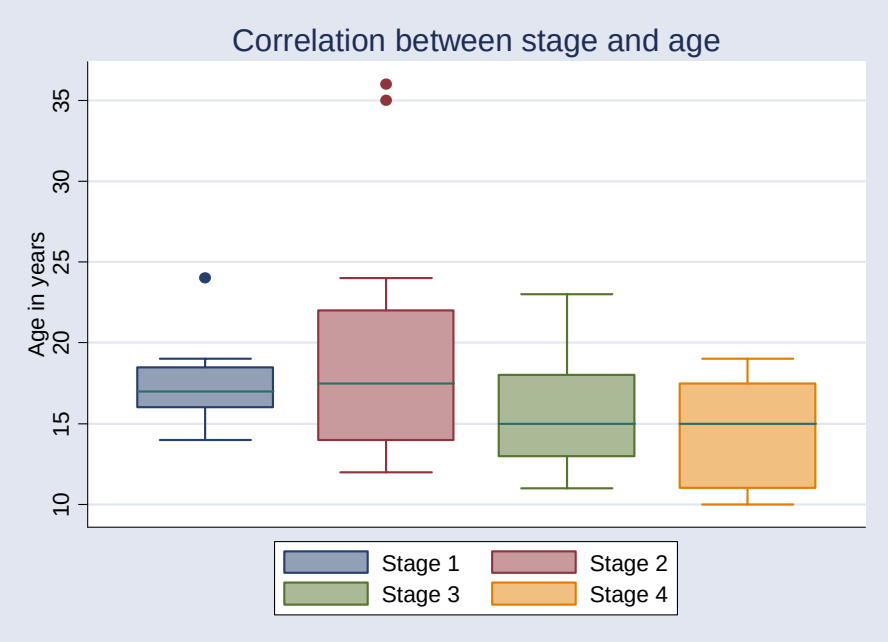


Table 6: Correlation between stage of disease and age

Stage of disease	Number of patients	Mean age in years	Standard deviation
Stage I	8	17.63	2.97
Stage II	22	19.05	6.40
Stage III	21	15.81	3.52
Stage IV	8	14.5	3.46

There was also a trend (P=0.099) towards a longer duration of epistaxis in patients with more advanced disease (Table 7). There was no association between the duration of nasal obstruction and stage of disease.

Table 7: Correlation between duration of epistaxis and nasal obstruction and disease stage

Stage of disease	Number of patients	Mean (SD) duration of epistaxis in months	Mean (SD) duration of nasal obstruction in months
Stage I	8	2.50 (2.33)	3.88 (3.23)
Stage II	22	7.73 (9.25)	7.77 (8.46)
Stage III	21	6.43 (5.96)	7.30 (6.79)
Stage IV	8	12.25 (10.62)	9.38 (6.76)

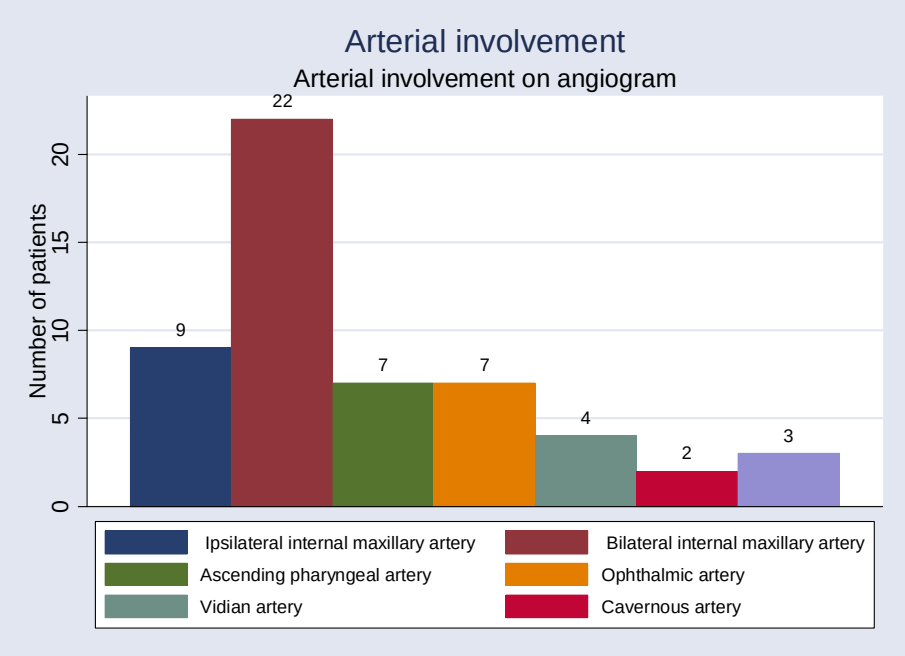
Arterial involvement on angiogram and embolization

In patients with primary disease, the branches of the external and internal carotid artery were found to supply the tumour (Table 8). In the 31 patients who underwent angiogram and embolization, the internal maxillary artery was the major feeding vessel in all cases. Bilateral internal maxillary artery was the most common arterial supply (70.96%). The tumour was also supplied by ascending pharyngeal artery in 7 cases. Further vascular supply from the internal carotid artery (branches of ophthalmic artery, vidian artery and cavernous artery) was evident (Figure 10).

Table 8: Arterial involvement on angiogram

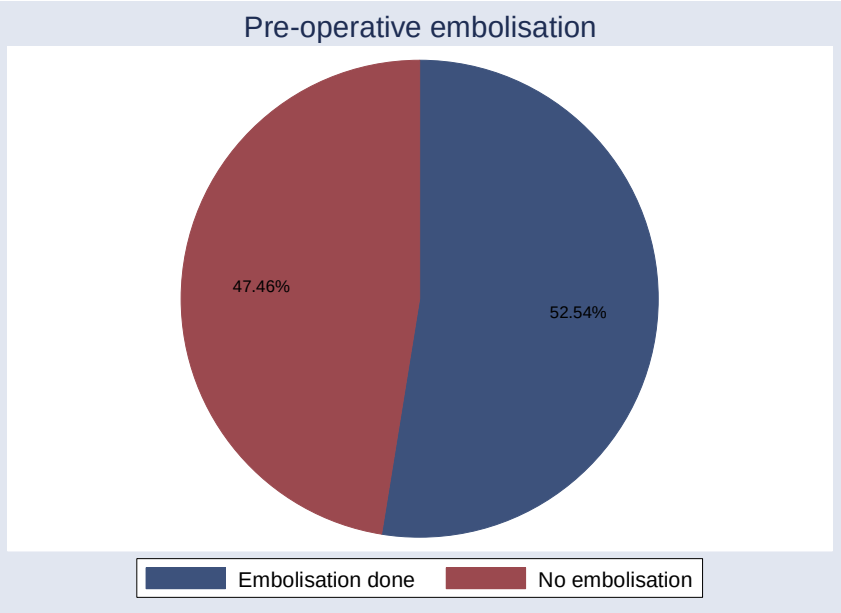
Artery	Branch involved	Number of patients	Percentage
External carotid artery	Ipsilateral internal maxillary artery	9	29.03
	Bilateral internal maxillary artery	22	70.96
	Ascending pharyngeal artery	7	22.58
Internal carotid artery	Ophthalmic artery	7	22.58
	Vidian artery	4	12.90
	Meningeal artery	2	6.45
	Cavernous artery	3	9.67

Figure 10: Arterial involvement on angiogram



Pre-operative embolization was performed in 31 patients (52.5%) before surgical resection (Figure 11). Branches of the external carotid artery were embolized whilst branches of the internal carotid artery were not embolized. Polyvinyl alcohol and gelform particles were used for embolization.

Figure 11: Pre-operative embolization



Surgical approaches

All 59 patients selected for the study group underwent surgical excision of the tumour. The transpalatal approach was the most frequently used surgical approach (Table 9).

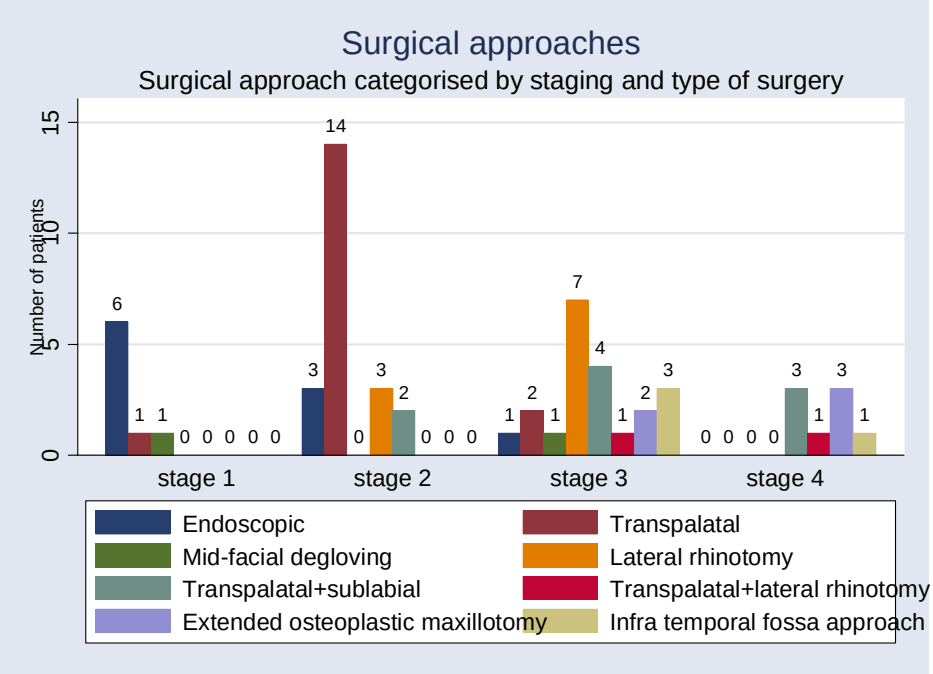
Table 9: Overview of surgical approach

Surgical approach	Frequency	Percent
Transpalatal	17	28.8
Endoscopic	10	16.9
Lateral rhinotomy	10	16.9
Transpalatal + sublabial	9	15.3
Extended osteoplastic maxillotomy	5	8.5
Infra temporal fossa approach	4	6.8
Midfacial degloving	2	3.4
Transpalatal + lateral rhinotomy	2	3.4
Total	**59**	**100**

When the surgical approach was categorized by staging, endoscopic surgery was the commonest treatment modality that was used in Stage I disease whilst transpalatal approach was the most frequent surgical approach for Stage II disease (Figure 12).

Different surgical approaches were used in Stage III disease whilst multiple surgical approaches were used for exposing the tumour in Stage IV disease (Figure 12).

Figure 12: Surgical approach categorized by staging



Complications

Comesis resulting after a Weber-Fergusson incision was acceptable in all patients. There was no per-operative mortality. The blood loss, as expected, was significantly higher (P=0.001) in patients with more advanced disease (Figure 13). There was no demonstrable effect of embolization on the volume of blood loss when patients were categorized according to the stage of disease and embolization (Figure 14). The mean (SD) blood loss was 660 (366) ml with the endoscopic approach (10 patients) and 685 (510) ml with the transpalatal approach (Table 10). There was a significantly higher blood loss (P=0.02) in patients who underwent transpalatal and sublabial surgery (1856 ± 1300 ml) compared with patients who underwent lateral rhinotomy (770 ± 519 ml).

Table 10: Type of surgical approach and blood loss

Surgical approach	No of patients	Mean blood loss (SD)	Range
Endoscopic	10	660 (366)	200 - 1300
Transpalatal	17	685 (510)	200 – 1800
Midfacial degloving	2	650 (71)	600 – 700
Lateral rhinotomy	10	770 (519)	300 – 2000
Transpalatal + sublabial	9	1856 (1300)	700 – 5000
Transpalatal + lateral rhinotomy	2	1200 (566)	800 – 1600
Extended osteoplastic maxillotomy	5	1900 (1575)	400 – 4000
Infra temporal fossa approach	4	1625 (479)	1000 - 2000

Figure 13: Blood loss correlated with staging of tumour

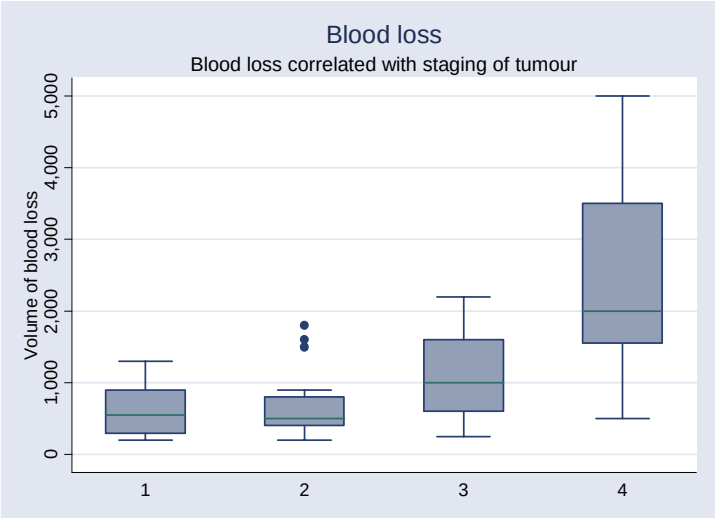
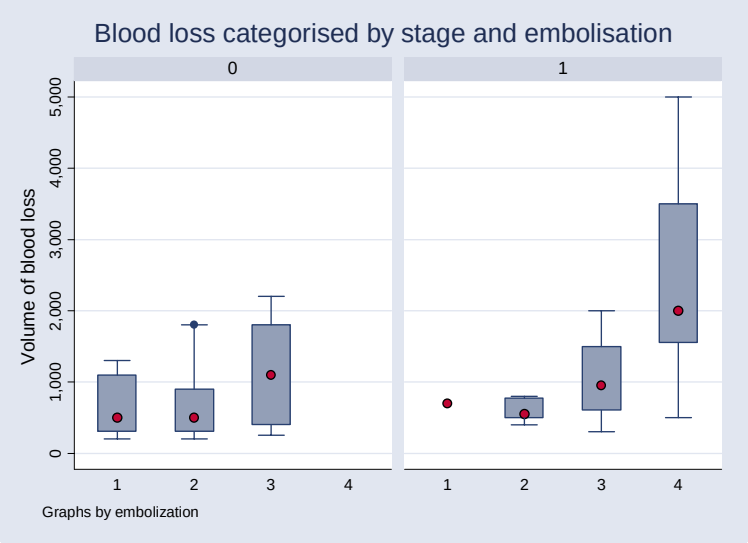
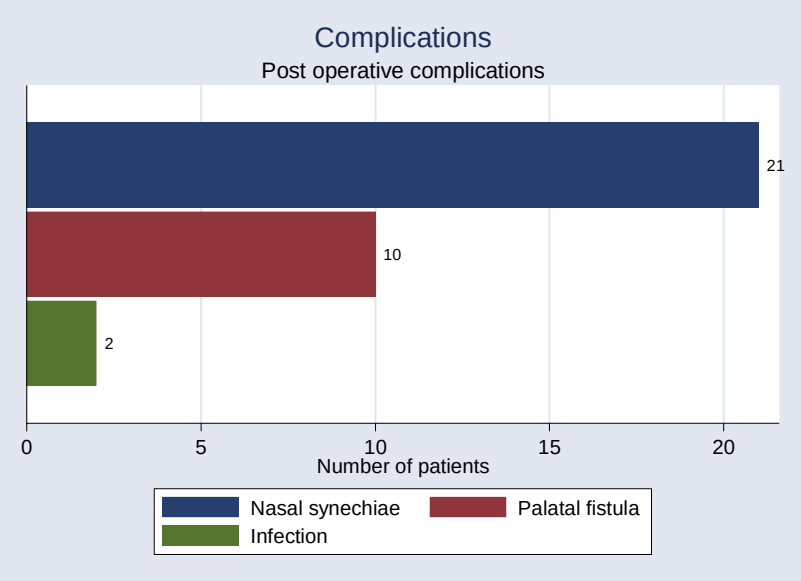


Figure 14: Blood loss categorized by stage and embolization



Other complications of surgery included nasal synechae in 22 patients, palatal fistula in 10 patients and infections in 2 patients (Figure 15).

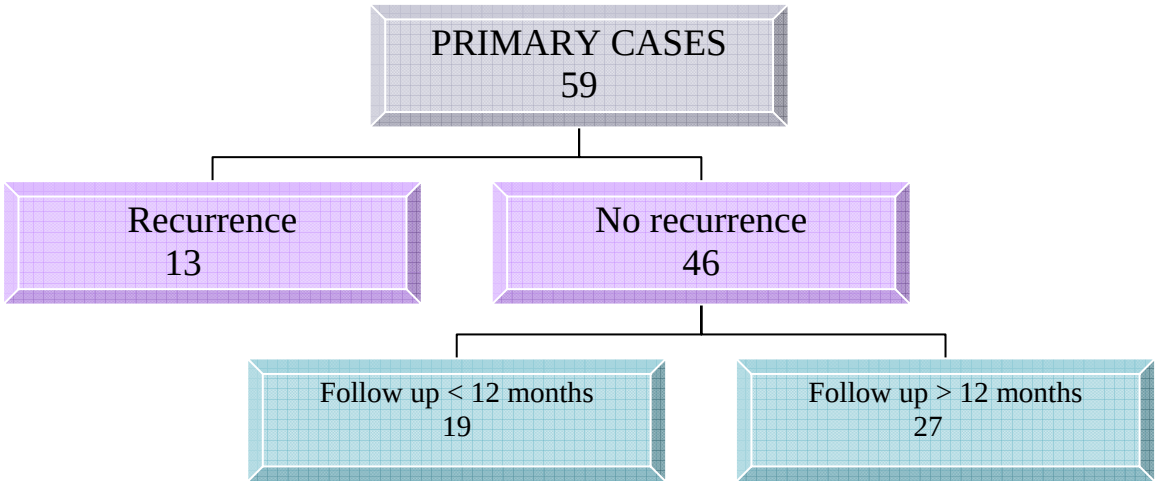
Figure 15: Complications



Recurrence

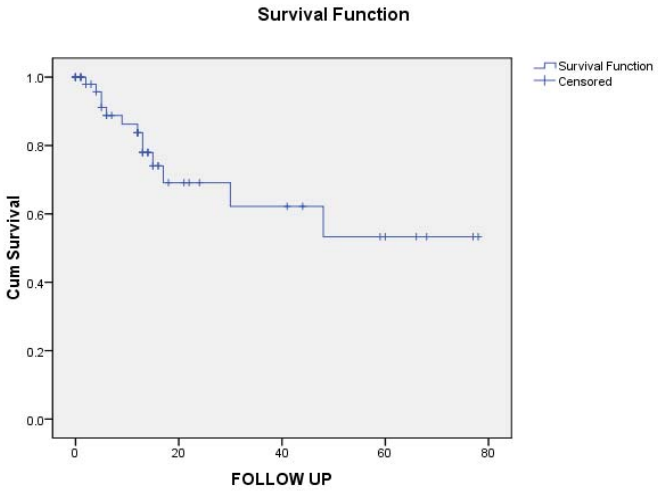
Of the 59 patients who presented with primary disease, 13 patients developed recurrence during the course of follow up. Of the 46 patients who did not have recurrence of disease, 19 patients had a follow up of less than 12 months (Figure 16).

Figure 16: Recurrence data



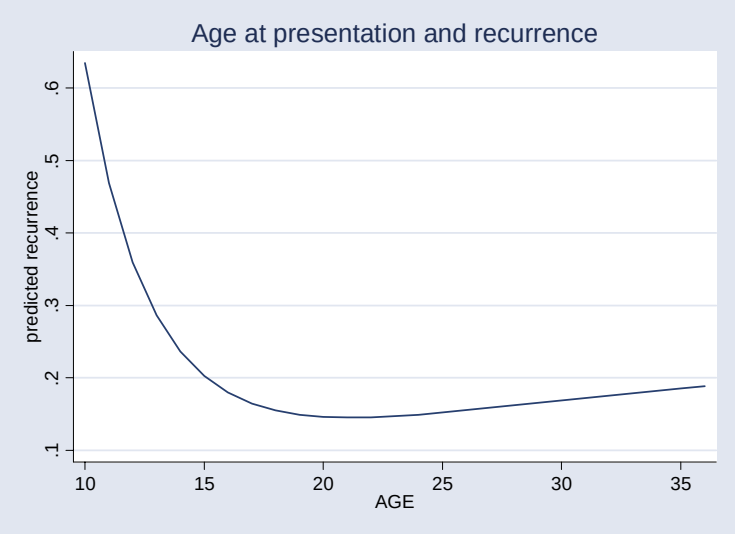
Recurrence seemed to occur primarily within the first 20 months of treatment for primary disease (Figure 17)

Figure 17: Survival analysis of recurrence



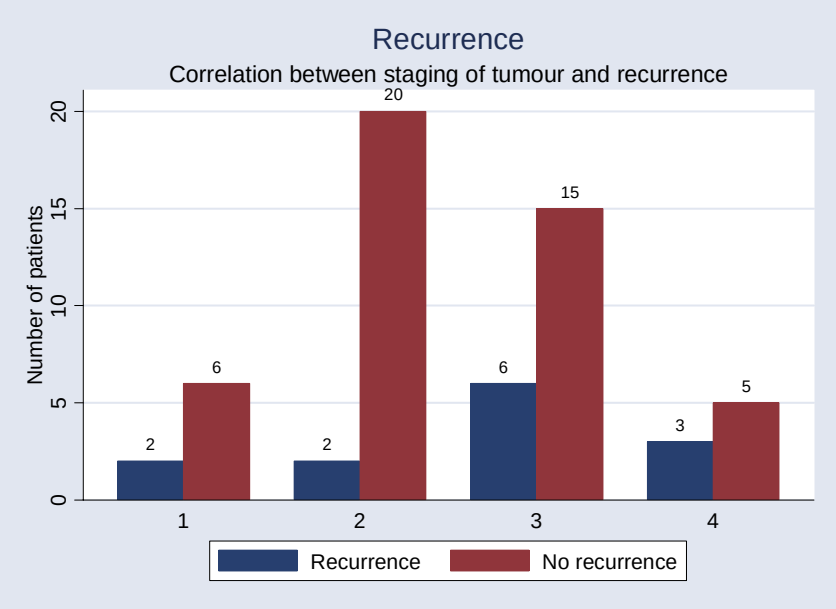
Recurrence tended to occur in younger patients although this did not reach statistical significance (Figure 18). A significant proportion of these (5/13, 38%) occurred within 6 months of surgery for primary disease. Since younger patients tended to present with more advanced disease, it is possible that the recurrence occurring in younger patient could possibly be a reflection of more advanced disease.

Figure 18: Correlation between age at presentation and recurrence



A higher proportion of patients with Stage III or Stage IV disease had recurrence (Figure 19). However this again did not reach statistical significance (P=0.16).

Figure 19: Staging of disease and recurrence



There was no association between pre-operative embolization and recurrence (6/28 versus 7/31; no embolization versus embolization, P=1.0). When the type of surgical procedure and recurrence rate was evaluated, it was found that recurrence rate was 10% with the endoscopic approach (1/10). The patient with recurrence had stage III tumour. None of the patients with stage I and II tumour undergoing the endoscopic approach had recurrence. The combined transpalatal and sublabial approach was associated with the highest (5/9, 55.6%) recurrence rates, followed by midfacial degloving (1/2, 50%) and infratemporal approaches (2/4, 50%). In contrast, the recurrence rate of 10% (1/10) in patients who underwent the lateral rhinotomy approach, trended to be lower (P=0.057) when compared with the combined transpalatal and sublabial approach. The rate of recurrence was 20% (1/5) with the extended osteoplastic maxillotomy approach which was used for advanced tumour (stage III and stage IV).

Table 11: Type of surgical approach and recurrence

Surgical approach	Number with recurrence (%)	Number without recurrence (%)
Endoscopic	1 (10)	9 (90)
Transpalatal	2 (11.8)	15 (88.2)
Midfacial degloving	1 (50)	1 (50)
Lateral rhinotomy	1 (10)	9 (90)
Transpalatal + sublabial	5 (55.6)	4 (44.4)
Transpalatal + lateral rhinotomy	0 (0)	2 (100)
Extended osteoplastic maxillotomy	1 (20)	4 (80)
Infra temporal fossa approach	2 (50)	2 (50)
Total	**13 (22.0)**	**46 (88.0)**

SECONDARY CASES

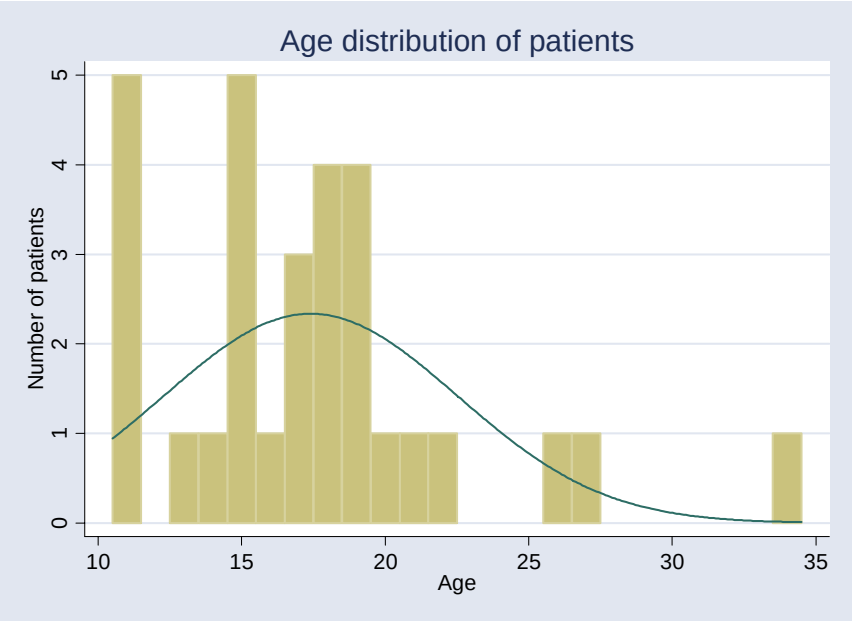
Demographics of secondary cases

The secondary cases comprised of 30 patients with a mean (SD) age of 17.4 (5.12) years and a range of 11 to 34 years (Figure 20). The majority (83.3%) were <20 years of age (Table 12). All patients were male.

Table 12: Age distribution of patients

Age group	Number of patients	Percentage
10-15 years	12	40
16-20 years	13	43.3
21-25 years	2	6.7
26-30 years	2	6.7
> 30 years	1	3.3
Total	**30**	**100**

Figure 20: Age distribution of patients (secondary cases of JNA)



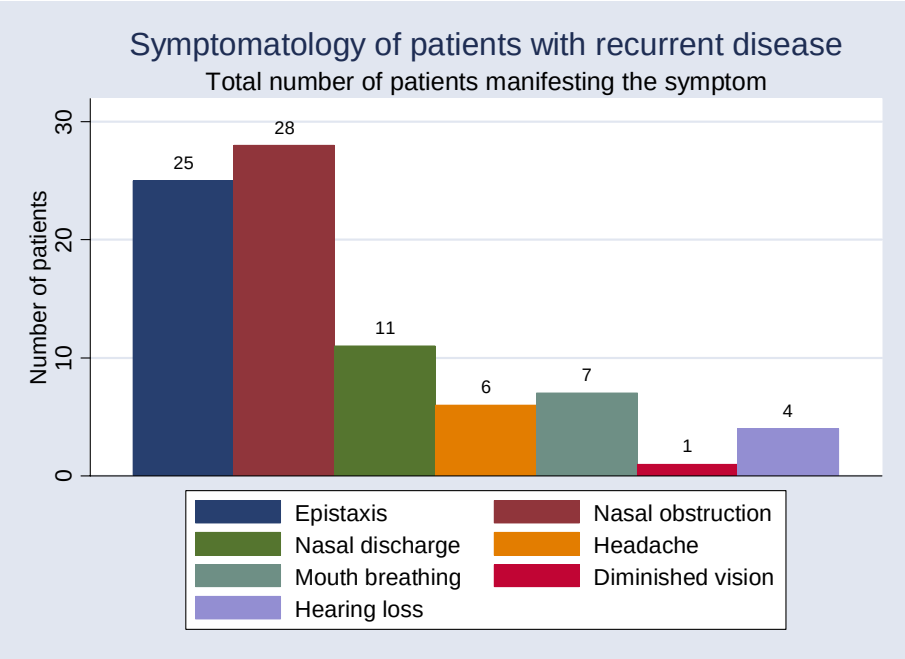
Symptomatology

The mean (SD) symptom free interval following previous surgery was 11.3 (10.82) months. The commonest symptoms that the patients with recurrent disease presented (Table 13) with were epistaxis (83.3%) and nasal obstruction (93.3%). Other symptoms included nasal discharge, headache, mouth breathing, diminished vision and hearing loss (Figure 21).

Table 13: Spectrum of symptoms of patients with recurrent disease

Symptom	Number of patients	Percentage
Nasal obstruction	28	93.33
Epistaxis	25	83.33
Nasal discharge	11	36.66
Mouth breathing	7	23.33
Headache	6	20
Hearing loss	4	13.33
Diminished vision	1	3.33

Figure 21: Symptomatology of patients with recurrent disease



The mean duration of symptoms prior to diagnosis of recurrence was 7-10 months for epistaxis, nasal obstruction, nasal discharge and headache (Figure 22). The duration of diminished vision and hearing loss were much longer (12 months and 10.5 months respectively) probably reflecting persistence of symptoms of patients with primary disease (Table 14). About 50% of patients with recurrent disease presented within 6 months of onset of epistaxis or nasal obstruction (Table 15).

Figure 22: Duration of symptoms in patients with recurrent disease

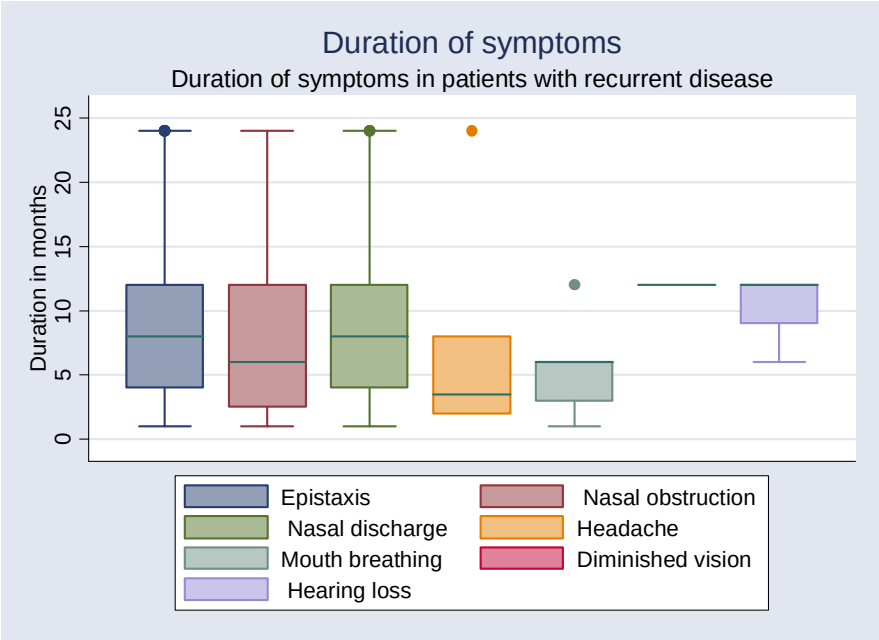


Table 14: Duration of symptoms in patients with recurrent disease

Symptom	No of patients (%)	Mean duration of symptom (months)	Standard deviation	Range (months)
Epistaxis	25 (83.3)	9.72	7.68	1 – 24
Nasal obstruction	28 (93.3)	8.89	7.61	1 – 24
Nasal discharge	11(36.33)	9.82	7.96	1 – 24
Headache	6 (20.0)	7.18	8.54	2 – 24
Mouth breathing	7 (23.33)	5.43	3.46	1 – 12
Diminished vision	1 (3.33)	12	NA	NA
Hearing loss	4 (13.33)	10.5	3.0	6 - 12

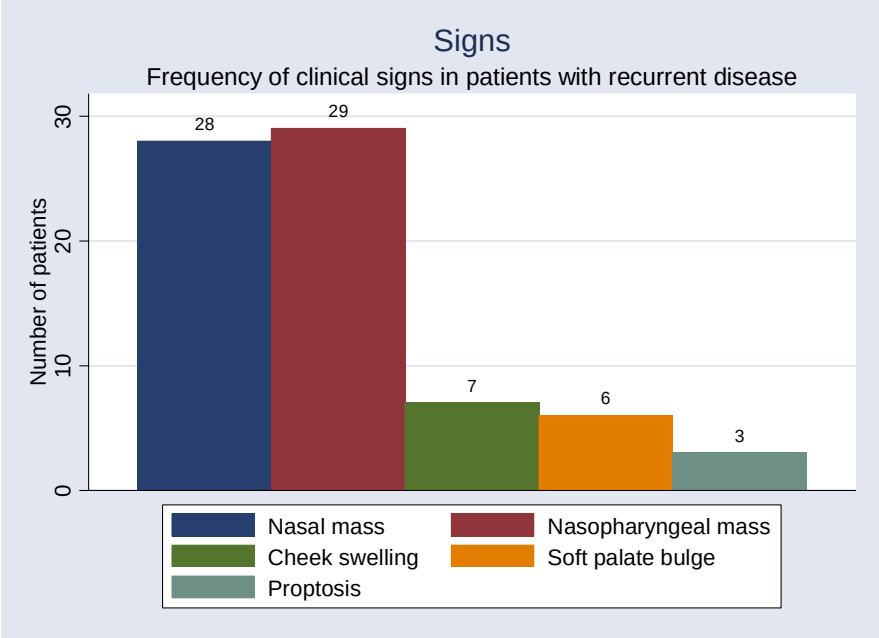
Table 15: Analysis of duration of epistaxis and nasal obstruction

Duration of symptom	No (%) of patients with epistaxis	No (%) of patients with nasal obstruction
Up to 1 month	4 (16)	6 (21.4)
1 to 6 months	7 (28)	9 (32.1)
6 to 12 months	9 (36)	8 (28.6)
> 12 months	5 (20)	5 (17.9)

Clinical Signs

On examination a nasal mass was seen in 28 (93.3%) patients whist a nasopharyngeal mass was seen in 29 (96.7%) patients (Figure 23). Cheek swelling was observed in 7 patients (23.3%), soft palate bulge in 6 patients (20%) and proptosis in 3 patients (10%).

Figure 23: Frequency of clinical signs



Extent of tumour

Pre-operative computed tomography (CT) scans were done in all 30 patients. The nasal cavity, nasopharynx and sphenopalatine regions were most commonly involved. The anatomic locations that the tumour involved are summarized in Table 16. Extradural and cavernous sinus involvement was seen in 8 patients (26.7%). As with the primary cases, the optic canal, foramen lacerum and pituitary fossa were not involved in any patient nor were intra-dural extension observed.

Table 16: Involved anatomic location of recurrent angiofibromas

Location *	Number	Percentage
Nasopharynx	29	96.7
Sphenopalatine region	29	96.7
Nasal cavity	28	93.3
Pterygoid palatine fossa	23	76.7
Base of pterygoid	19	63.3
Sphenoid sinus	19	63.3
Infratemporal fossa	16	53.3
Ethmoid sinus	11	36.7
Inferior orbital fissure	10	33.3
Pterygoid plate	8	26.7
Interpterygoid fossa	8	26.7
Cavernous sinus	8	26.7
Extradural extension	8	26.7
Foramen rotundum	7	23.3
Parapharyngeal space	6	20
Superior orbital fissure	4	13.3
Orbital apex	4	13.3
Maxillary sinus	3	10
Foramen ovale	1	3.3
Clivus	1	3.3

* None of the patients had involvement of the optic canal, foramen lacerum, pituitary fossa, anterior cranial fossa or intradural extension

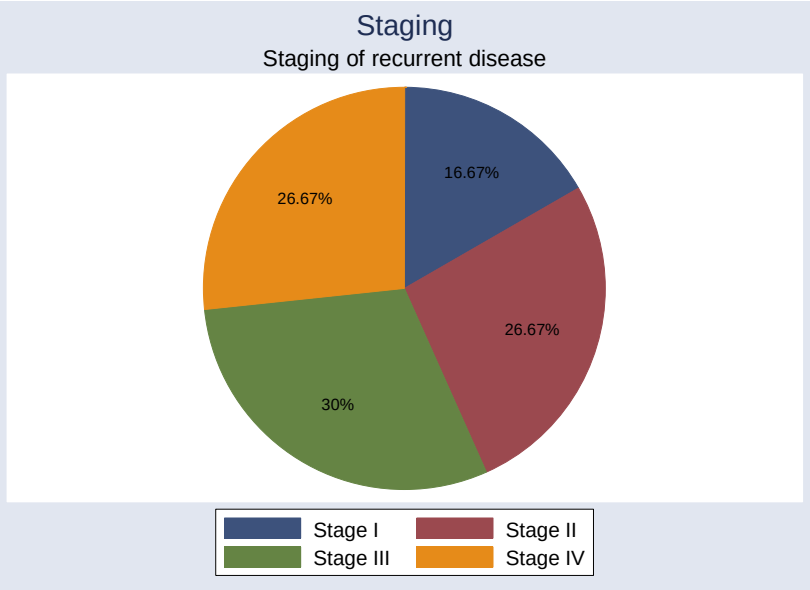
Staging

Stage I disease was seen in 16.7% of patients who presented with recurrence. The majority of the cases (Figure 24) operated were Stage II (26.7%), Stage III (30%) and Stage IV (26.7%). Unlike primary cases, there was no association between age and the stage of disease. There was also no correlation between the duration of epistaxis or nasal obstruction and the stage of disease (Table 17).

Table 17: Correlation between age, duration of epistaxis and nasal obstruction and stage of disease

Stage of disease	Number of patients	Mean (SD) age in months	Mean (SD) duration of epistaxis in months	Mean (SD) duration of nasal obstruction in months
Stage I	5	17.20 (3.96)	5.20 (10.55)	7.80 (9.34)
Stage II	8	18.88 (5.94)	10.50 (7.01)	10.50 (7.01)
Stage III	9	17.00 (7.02)	6.67 (8.34)	5.89 (8.48)
Stage IV	8	16.50 (2.14)	9.12 (7.06)	9.12 (7.06)

Figure 24: Stage of disease



Angiogram and embolization

Eleven patients (36.7%) underwent pre-operative angiogram and embolization before surgery. As with primary cases, bilateral internal maxillary artery was the commonest arterial supply (63.63%). The other feeding vessels are summarized in Table 18. Again as with primary cases, only the branches of the external carotid artery were embolised.

Table 18: Arterial involvement of secondary cases on angiogram

Artery	Branch involved	Number of patients	Percentage
External carotid artery	Ipsilateral internal maxillary artery	4	36.36
	Bilateral internal maxillary artery	7	63.63
	Ascending pharyngeal artery	2	18.18
Internal carotid artery	Ophthalmic artery	0	0
	Vidian artery	0	0
	Meningeal artery	1	9.09
	Cavernous artery	0	0

Surgical approaches

Thirty patients underwent surgical excision of the tumour. The transpalatal approach was the most frequently used surgical approach (Table 19).

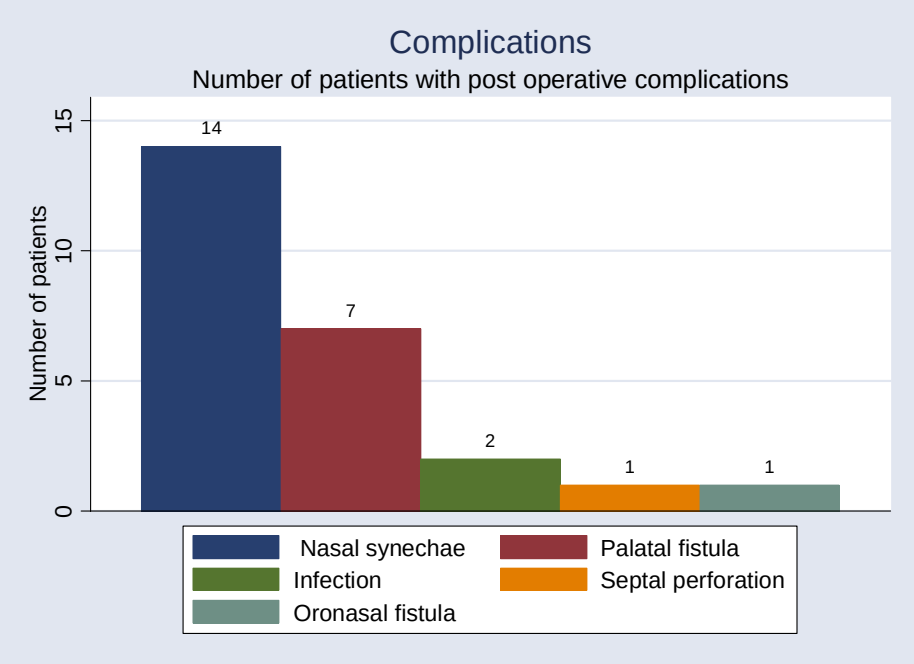
Table 19: Overview of surgical approach of secondary cases

Surgical approach	Frequency	Percent
Transpalatal	8	26.67
Lateral rhinotomy	6	20.0
Transpalatal + sublabial	4	13.33
Extended osteoplastic maxillotomy	4	13.33
Endoscopic	3	10.0
Infra temporal fossa approach	3	10.0
Midfacial degloving	2	6.67
Total	**30**	**100**

Complications

There was no operative mortality. The blood loss was significantly higher with Stage IV disease (1988 ± 1255 ml) compared with either Stage I (590 ± 219) or Stage II or III disease (Mean 800-850 ml). There was no difference in blood loss between embolized and non-embolized patients or with type of surgery. Nasal synechae developed in 46.7% of patients. Other complications included palatal fistula in 7 patients (23.3%), infection in 2 patients and septal perforation and oronasal fistula in 1 patient each (Figure 25).

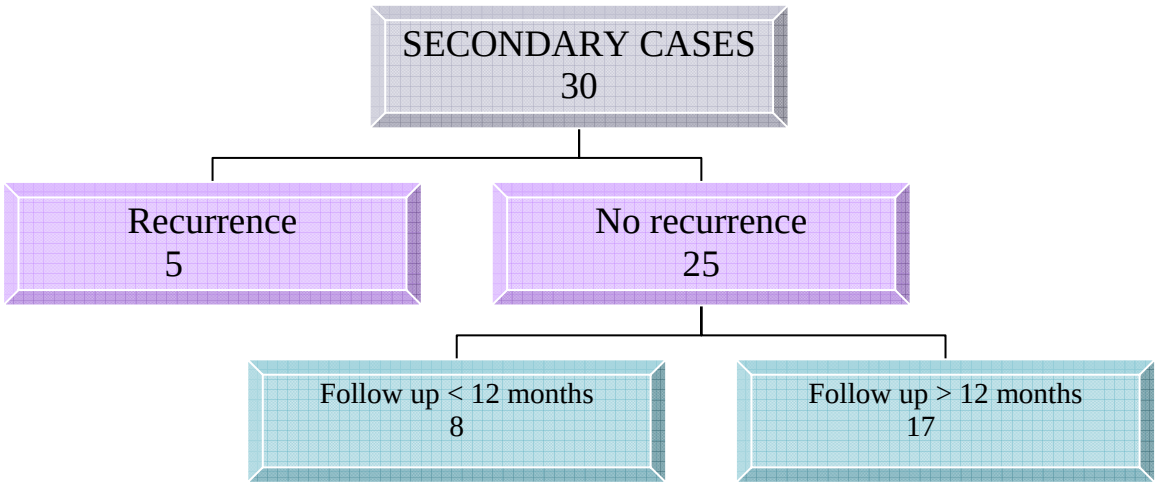
Figure 25: Complications



Recurrence

Of the 30 patients in the secondary disease cohort, 5 patients had further recurrence after surgical treatment (Figure 26). Of the 25 patients who did not have recurrence 8 patients were followed up for < 12 months. The mean (SD) duration of follow up of the secondary cases was 18.77 (23.79) months.

Figure 26: Recurrence data of secondary cases



There was no correlation between age of the patient and recurrence (P=0.5) nor was there any association between recurrence and the duration of epistaxis (P=0.15) or nasal obstruction (P=0.35). There were no further recurrences in patients who were treated for recurrent Stage I disease. The recurrence rates were higher in Stage IV disease (3 out of 5 patients) although a statistically significant association between the stage of disease and recurrence was not evident (P=0.47). There were no differences in the recurrence rates amongst different surgical approaches.

DISCUSSION

In this study we analyzed the clinical profile and the rate of recurrence of juvenile nasopharyngeal angiofibroma in patients who presented to our institution between 1998 and 2008. There have been few reports of this disease from India[14, 33, 34, 35] and this series would add to existing knowledge on JNA. In addition, this cohort contains patients who were treated by endoscopic surgery, a relatively new therapeutic option for this disease. We have also systematically analyzed patients with recurrent disease and have attempted to elucidate the factors that may contribute to recurrence.

Symptomatology and signs

The primary cohort consisted of patients of a young age group with a mean (SD) age of 17.08 (4.96) years. This was very similar to other cohorts from India (Table 20). The commonest symptoms at presentation were epistaxis and nasal obstruction. This was again similar to other cohorts from India. Nasal mass was observed in all our patients whereas other studies have reported variable occurrence from 60-100%. A nasopharyngeal mass was almost universally seen in all series.

Cheek swelling was seen only in 28.8% of our patients whilst another study from India[35] has reported this manifestation in 57.9% of patients. The incidence of proptosis was again much lower in our cohort (5.1%) compared with the cohort by Tyagi et al[35] where the incidence was 21%.

Table 20: Comparison of demographics, symptoms and signs of primary cases
Current study versus other Indian studies

Parameter	Current study	Malik et al[33]	Mistry et al[34]	Tandon et al[14]	Tyagi et al[35]
Number of primary cases	59	27	32	37	95
Number of recurrent cases	30	NA	NA	13	NA
Mean (SD) age of primary	17.08 (4.96)	15.5 (NA)	16 (NA)	15.9 (NA) †	NA
Epistaxis (%)	96.6	96.3	NA	90	97.9
Nasal obstruction (%)	91.5	92.6	NA	88	100
Nasal mass (%)	100	88.9	NA	60†	89.4
Nasopharyngeal mass (%)	100	100	NA	94 †	100
Cheek swelling (%)	28.8	22.2	NA	10	57.9
Soft palatal bulge (%)	20.3	NA	NA	NA	31.5
Proptosis (%)	5.1	3.7	NA	14	21

† Data for the entire cohort of 50 patients

When we compare our data with Western data, we again find that the mean age of presentation of our patients was similar to other Western cohorts (Table 21). The incidence of epistaxis and nasal obstruction were higher in our cohort of patients (>90%) as compared with Western data (68-85%). One study from Egypt[6] noted a high incidence of epistaxis (95%) comparable with our data (96.6%). Data on clinical signs were sparse in Western studies and hence could not be compared.

Table 21: Comparison of demographics, symptoms and signs of primary cases
Current study versus Western studies

Parameter	Current study	Pryor et al[5]	Hardillo et al[12]	El-Banhawy et al[6]	Economou et al[36]
Number of primary cases	59	58	29	20	83
Number of recurrent cases	30	7	NA	NA	NA
Mean (SD) age of primary	17.08 (4.96)	15	17.9 (NA)	15.5 (NA)	17(NA)
Epistaxis (%)	96.6	85	79.31	95	73
Nasal obstruction (%)	91.5	68	82.8	75	71
Nasal mass (%)	100	NA	NA	NA	NA
Nasopharyngeal mass (%)	100	NA	NA	NA	NA
Cheek swelling (%)	28.8	NA	NA	NA	NA
Soft palatal bulge (%)	20.3	NA	NA	NA	NA
Proptosis (%)	5.1	14	NA	NA	7

Surprisingly duration of symptoms was higher in secondary cases when compared to primary cases. Mean duration of alarming symptom like epistaxis was 9.7 months in secondary cases. Reasons for this are unclear and this need to be further studied.

Extent of tumour

The nasal cavity and nasopharynx were involved in all our patients. In a study done by Tyagi et al[35] the nasopharynx was involved in 100% and nasal cavity in 89.4%. The sphenopalatine region, sphenoid sinus and pterygopalatine fossa were involved in more than 50% of our patients. None of our patients had involvement of the optic canal, foramen lacerum, pituitary fossa or intradural extension.

Intracranial extension was seen in 16.9% and 26.7% patients in primary cases and secondary cases respectively. Economou et al[36] reported intracranial extension in 21% of patients. About 50% of our patients presented with advanced disease (Stage III or IV). Comparison of staging of disease with other Indian cohorts was made difficult by the fact that several other staging systems were used. In a comparable cohort that used the Fisch classification, advanced disease was observed in 33% of patients.[37] In another study by Andrews et al,[18] 30% of patients presented with advanced disease.

In primary cases, we observed that there was a trend towards younger patients presenting with more advanced disease but there was no correlation between age and stage in secondary cases. An association between age and stage of disease has been demonstrated in other case series. Tandon et al[14] observed that tumours tended to grow slower in patients who are in their late teens or early twenties, than in patients of 15 years or

younger. In primary cases there was also a trend towards a longer duration of epistaxis in patients with more advanced disease, but no such trend seen in secondary cases.

Arterial involvement on angiogram

In the current study, the internal maxillary artery was the major feeding vessel in all patients who underwent an angiogram. An interesting aspect in our study was that bilateral internal maxillary arterial supply to the tumour was more commonly observed (71%) than ipsilateral internal maxillary arterial supply (29%). In contrast, Nicoli et al[37] in their cohort of 15 patients observed that 73.4% of patients had strictly unilateral vascular supply and only 26.6% had contralateral feeding vessels.

Surgical approaches

A range of surgical approaches were used in the management of JNA, reflecting a diverse approach by a large team of surgeons as well as changing surgical trends over time. It is interesting to note that our cohort had 10 patients who were treated with the more recently described endoscopic surgical approach. Small cohorts of endoscopically treated JNA are described in literature. Roger et al[27] have treated 20 cases with purely endoscopic resection. In our small cohort of patients, one of the 10 patients (10%) had recurrence of disease, 6 months after the initial procedure.

The traditional transpalatal approach was the commonest approach that was used in our patients with Stage II disease. Extensive surgical approaches were used for advanced stage disease. The type of surgical approach was based on several factors that included

(a) the stage of tumour, (b) age of the patient (growth of craniofacial skeleton) (c) morbidity of the approach and (d) the tendency of tumour to become involuted or stabilized with maturity.[35]

Blood loss

In our study, the mean (SD) blood loss was not different between primary and secondary cases (1057 (909) versus 1095 (916) ml). In the study by Tandon et al,[14] the mean blood loss was 1057 ml in 50 patients. In our study, the patients who underwent endoscopic surgery, had a mean blood loss of 660 ml (n=10) and 733 ml (n=3) for primary and secondary cases respectively. In a study by Onerci et al,[30] the mean blood loss was 1000 ml in 8 patients operated by the endoscopic approach. Roger at el[27] reported a much lower blood loss of 350 ml in 20 patients undergoing endoscopic surgery. The mean blood loss with the transpalatal approach was similar between our cohort and the study reported by Tandon et al[14] (685 ml versus 843 ml). In our study, a significantly higher blood loss (P=0.02) was noted in patients who underwent transpalatal and sublabial surgery (1856 ± 1300 ml) compared with patients who underwent lateral rhinotomy (770 ± 519 ml). The blood loss was significantly higher in patients with more advanced disease (Figure 13). Similarly Radkowski et al[11] found that there was a noticeable trend toward increased blood loss with increasing preoperative stage.

There was no demonstrable effect of embolization on the volume of blood loss in both primary and secondary cases. A similar effect was noted by Petruson et al[2] who observed that pre-operative embolization did not impact blood loss (1300 versus 1250 ml,

embolized versus non-embolized group respectively). Glad et al[7] found that preoperative embolization significantly decreased the intraoperative blood loss (mean blood loss was 650ml in embolized group compared to 1200 ml in non embolized group. It is unclear at this stage if pre-operative embolization reduces intra-operative blood loss in patients with JNA.

Recurrence

In our study, the recurrence rate was 22% in primary cases and 16.7% for secondary cases. The mean (SD) duration of follow up were 16.95 (20.32) and 18.77 (23.79) months for primary and secondary cases respectively. Recurrence seemed to occur primarily within the first 20 months of treatment for primary cases. The recurrence rates are similar to other published cohorts (Table 22)

Table 22: Recurrence rates

	Rate of recurrence in primary cases	Rate of recurrence in secondary cases
Current study	22%	16.7%
Herman et al[19]	27.5%	NA
Mann et al[8]	25%	40%
Radkowski et al[11]	22%	NA

In our study, recurrence tended to occur in the younger patients (Table 13) and this is similar to observations by Petruson et al.[2] It is possible that recurrence in younger patients is a reflection of more advanced disease in younger patients. The presence or duration of any of the symptoms or signs did not predict recurrence.

Table 23: Univariate analysis of factors that predict recurrence

Continuous variables	Patients with recurrence (n=13)		Patients without recurrence (n=46)		P value
	Mean	SD	Mean	SD	
Age (years)	15.38	3.66	17.56	5.20	0.05*
Duration of epistaxis (months)	7.38	9.04	7.43	7.84	0.51
Duration of nasal obstruction (months)	6.61	5.90	8.22	7.47	0.78
Stage of tumour	2.77	1.01	2.41	0.86	0.13

Discrete variables	Patients with recurrence (n=13)	Patients without recurrence (n=46)	P value
History			
Epistaxis	13	44	1.0
Nasal obstruction	13	41	0.57
Nasal discharge	8	25	0.76
Mouth breathing	3	13	1.0
Headache	6	8	0.06
Decreased vision	2	1	0.12
Hearing loss	0	4	0.57
Signs			
Nasal/NP mass	13	46	1.0
Cheek swelling	5	12	0.49
Palatal bulge	3	9	0.72
Proptosis	2	1	0.12

The pre-operative tumour stage was not found to be significantly associated with recurrence in our cohort of patients. Radkowski et al[11] found that preoperative tumour stage was the primary factor affecting tumour recurrence.

Some studies have reported increased risk of recurrence with pre-operative embolization. Lloyd et al[31] reported that embolization was a contributory cause of recurrence as it shrinks the tumour making total removal more difficult especially if there is deep invasion of sphenoid, and concluded that patients who showed this on preoperative CT are at high risk of recurrence and embolization is contraindicated. In the study by Petruson et al,[2] 7 patients (41%) who were treated with preoperative embolization had a

recurring tumour compared to only 1 patient (8%) in the non-embolized group. We did not however note an association between embolization status and recurrence.

The association between the choice of surgical approach and recurrence rates is important. Analysis of recurrence is mandatory to validate the choice of any surgical approach, but this is difficult because JNA is a rare disorder. In our study, with the endoscopic approach only 1 patient had recurrence out of 10. This patient had stage III tumour. None of the patients with stage I and II tumour who were operated with the endoscopic approach had recurrence. These results indicate that endoscopic approach is as effective as open surgery for early stage tumour. Possibly for late stage tumours endoscopic approach may not be ideal as resection may not be complete and technically difficult.

Onerci et al[30] used endoscopic technique in early and late stage (minimal intracranial extension) angiofibroma. They were able to excise tumour completely in early stage tumour. However some residual tumour was observed in late stage tumour. They suggested that in late stage tumour, the surgeon must be prepared to shift or use combined technique, otherwise there is a possibility for extensive residual lesion. Lateral tumour extension to cavernous sinus or optic nerve, extensive extension to middle cranial fossa and posterior to pterygoid plates are definite limits for endoscopic surgery.

In our cohort, the combined transpalatal and sublabial approach was associated with the highest (5/9, 55.6%) recurrence rates, followed by midfacial degloving (1/2, 50%) and

infratemporal approach (2/4, 50%). In contrast the recurrence rate was only 10% (1/10) in patients who underwent the lateral rhinotomy approach. Similar results were reported by Radkowski et al[11] in which the combined transpalatal and transantral approach was associated with a 75% recurrence rate compared with a 9.1% recurrence rate with lateral rhinotomy approach. The rate of recurrence was 20% (1/5) in our study with extended osteoplastic maxillotomy approach which was used for advanced tumour (stage III and stage IV).

Tyagi et al[35] found that extension into the pterygoid fossa and basisphenoid, erosion of the clivus, intracranial extension medial to cavernous sinus, invasion of sphenoid dipole through a widened pterygoid canal were associated with an increase risk of recurrence. In our study we did not found any significant association of these areas and recurrence.

CONCLUSIONS

* The most common presenting symptoms were epistaxis and nasal obstruction in both primary and secondary cases.

* More advanced disease was observed in younger patients and in those who had a longer duration of epistaxis.

* Intra-operative blood loss was significantly higher in patients with more advanced disease.

* There was no effect of pre-operative embolization on intra-operative blood loss.

* The blood loss was significantly higher in patients who underwent surgery by the combined transpalatal and sublabial approach compared with the lateral rhinotomy approach.

* The mean (SD) symptom free interval following previous surgery was 11.3 (10.82) months in secondary cases.

* Younger patients had a tendency for recurrence.

* There was no association between embolization and recurrence in both primary and secondary cases.

* The rate of recurrence was higher with the combined transpalatal and sublabial approach when compared to the lateral rhinotomy approach.

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Informed Consent form to participate in an observational study

Study Title: "Clinical profile of Juvenile nasopharyngeal angiofibroma and analysis of recurrences"

Study Number:

Subject's Initials: _____ Subject's Name: _____
Date of Birth / Age:_____

Please initial box (Subject)

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]

(ii) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [ ]

(iii) I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published. [ ]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s) [ ]

(v) I agree to take part in the above study. [ ]

Date

Signature (or Thumb impression) of the Subject/Legally Acceptable Representative

Signatory's Name:

Date

Signature of the investigator
Study investigators name:

Informed Consent form to participate in an observational study
(For paediatric patient)

Study Title: "Clinical profile of Juvenile nasopharyngeal angiofibroma and analysis of recurrences"

Study Number:

Subject's Initials: _____ Subject's Name: _____
Date of Birth / Age:_____

Please initial box (Subject)

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]

(ii) I understand that my child's participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without his medical care or legal rights being affected. [ ]

(iii) I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my child's identity will not be revealed in any information released to third parties or published. [ ]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s) [ ]

(v) I agree that my child can take part in the above study. [ ]

Date

Signature (or Thumb impression) of the Subject/Legally Acceptable Representative/Guardian

Signatory's Name:

Date

Signature of the investigator
Study investigators name:

Patient Information sheet

Study Title:

Clinical profile of Juvenile nasopharyngeal angiofibroma (nasal tumour) and analysis of recurrences

Purpose of research:

1. To study the symptoms and signs of patients diagnosed to have this nasal tumour
2. To assess factors that may have contributed to recurrence of tumour

Expected duration of the Subject's participation:

Duration of study will be 18 months.

Description of the procedures:

This study would not involve any additional procedures or treatments

However treatments that are required as part of your regular follow up, such as scans and endoscopy, may be required and advised by your surgeon irrespective of whether you are part of the study or not. This would be determined by your treating surgeon.

Risks or discomforts to the Subject:

As the study does not include any trial treatment, there is no extra risk for the patient due to participation in study and there will not be any additional cost of treatment for the patient.

Benefits to the Subject:

Since you have presented for an evaluation by the ENT surgeon, the surgeon might be able to pick up any evidence of residual disease or recurrence. However if you have come for your routine follow up, there is no additional benefit to you.

Benefits to others:

The potential benefit for others is what new knowledge that we might gain in studying all the factors that might predict recurrence.

Confidentiality:

Patient's identity will not be revealed in any information or released to third parties or published.

Participation:

Your participation in the study is entirely voluntary and the patient is free to withdraw at any time, without giving any reason. Refusal to participate will not involve any penalty or loss of benefits to which the Subject is otherwise entitled.

Contact person:

Dr Sunil Jalan, Dept of E.N.T., CMCH, Vellore

PROFORMA

NAME HOSPITAL NO.

AGE SEX

ADRESS

PHONE NO.

DATE OF ADMISSION

DATE OF SURGERY

DATE OF DISCHARGE

HISTORY OF PREVIOUS SURGERY YES (1) NO (2)
IF YES DATE OF SURGERY

SYMPTOM FREE INTERVAL FOLLOWING SURGERY (IN MONTHS) -

SURGICAL APPROACH

Endoscopic	1
Transpalatal	2
Lateral rhinotomy	3
Mid facial degloving	4
Extended osteoplastic maxillectomy	5
Infratemporal fossa approach	6
Endoscopic assisted	7
other	8

Symptoms	**YES (1)**	**NO (2)**	**IF YES Duration (In months)**
Epistaxis			
Nasal obstruction			
Nasal discharge			
Obligatory mouth breathing			
Headache			
Otalgia			
Dysphagia			
Blindness			
Hearing loss			

Signs	**YES (1)**	**NO (2)**
Nasal mass		
Nasopharyngeal mass		
Cheek swelling		
Bulged soft palate		
Proptosis		

Investigations-
HB - GM%

RIGID NASAL ENDOSCOPY: If done (1) Not done (2)
If done
Nasal cavity (1) nasopharynx (2)

CT SCAN –

Location	Yes(1)	No(0)
Nasal cavity		
Nasopharynx		
Sphenopalatine region		
Sphenoid sinus		
Pterygoid palatine fossa		
Infratemporal fossa		
Ethmoid sinus		
Base of pterygoid		
Inferior orbital fissure		
Pterygoid plate		
Orbital apex		
Extradural extension		
Maxillary sinus		
Foramen rotundum		
Cavernous sinus		
Interpterygoid fossa		
Parapharyngeal space		
Superior orbital fissure		
Foramen ovale		
Anterior cranial fossa		
Clivus		

CT STAGING

STAGE I	1
STAGE II	2
STAGE III	3
STAGE IV	4

Angiography done (1) not done (2)
If done
Supplying vessel
Int maxillary artery (1) ascending pharyngeal artery (2)

Embolization YES (1) NO (2)
If yes
Material used for embolization
Gel (1) Alcohol (2)

Surgical approach

Endoscopic	1
Transpalatal	2
Mid facial degloving	3
Lateral rhinotomy	4
Transpalatal+ Sublabial	5
Transpalatal+ Lateral rhinotomy	6
Extended osteoplastic maxillectomy	7
Infratemporal fossa approach	8

Intraop blood loss …….. ml

Duration of surgery: ……. hrs

Intraop blood transfusion YES (1) NO (2)
IF YES ……ML

Postop blood transfusion YES (1) NO (2)
IF YES ……ML

Duration of stay in surgical ICU --- Days

Duration of total in patient stay ……. Days

Anterior nasal pack removal …. Post op day

Posterior nasal pack removal ……. Post op day

Complication	YES (1)	NO (2)
Palatal fistula		
Wound infection		
Nasal synechae		
CSF leak		
Mal occlusion		
Residual disease		

Follow up
Follow up period …. Months

Recurrence YES (1) NO (2)
IF yes
SYMPTOM FREE INTERVAL FOLLOWING SURGERY (IN MONTHS) –

NO	H.NO	DOS	RECURRENC	AGE	EPISTAXIS	D OF EPI	NASAL OB	D OF NO	NASAL DIS	D OF ND	HEAD	D HEAD	M B	D OF MB	VIS	D OF V	HL	D OF HL	NAS	NPX	CHE	SP	PRO	STA	EMB	APR	BLO	SYN	FIST	INF
1	031300D	20/06/2007	0	35	1	5	1	5	1	5	0	0	0	0	0	0	0	0	1	1	0	0	0	2	1	4	500	0	0	0
2	837122C	08/10/2006	1	11	1	8	1	5	0	0	0	0	0	0	0	0	0	0	1	1	1	1	1	4	1	7	4000	0	1	1
3	080434C	23/10/2001	0	12	1	6	1	6	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	2	0	2	400	0	0	0
4	852268C	07/11/2006	0	18	1	1	1	2	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	1	0	1	300	0	0	0
5	257706C	21/02/2003	0	14	1	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	2	0	2	200	1	0	0
6	642674B	08/03/1998	1	22	1	6	1	6	1	6	0	0	0	0	0	0	0	0	1	1	0	0	0	3	0	5	700	0	0	0
7	940359B	01/10/2000	0	15	1	8	1	8	0	0	0	0	0	0	0	0	0	0	1	1	1	1	0	4	1	5	1500	0	0	0
8	190856D	08/01/1900	0	17	1	7	1	7	1	7	0	0	0	0	0	0	0	0	1	1	1	0	0	3	0	4	400	0	0	0
9	283606D	10/09/2008	0	17	1	10	1	10	1	4	0	0	0	0	0	0	0	0	1	1	0	0	0	3	1	4	500	0	0	0
10	928021B	03/07/2002	0	19	1	8	1	6	1	6	0	0	0	0	0	0	1	1	1	1	1	0	0	4	1	6	1600	1	0	0
11	992544B	03/09/2001	1	13	1	3	1	4	1	3	0	0	0	0	1	3	0	0	1	1	1	0	0	3	1	8	1500	0	0	0
12	742758B	06/11/1999	1	14	1	6	1	6	1	6	1	6	0	0	1	2	0	0	1	1	0	1	1	3	1	4	1200	1	0	0
13	617396C	04/07/2005	0	20	1	2	1	3	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	3	1	4	1000	1	0	0
14	311077C	06/04/2003	0	14	1	8	1	8	0	0	1	8	1	8	0	0	0	0	1	1	0	0	0	2	0	2	1800	0	0	0
15	945376C	17/01/2007	0	14	1	24	1	24	1	12	1	12	1	24	0	0	0	0	1	1	0	0	0	3	1	7	1600	1	0	0
16	985194B	02/02/2001	1	18	1	5	1	5	1	5	1	4	0	0	0	0	0	0	1	1	0	0	0	2	0	2	200	1	1	0
17	639751c	06/10/2005	0	16	1	2	1	2	1	2	0	0	0	0	0	0	0	0	1	1	0	0	0	1	0	1	1100	0	0	0
18	310594C	07/01/2003	0	24	1	36	1	36	0	0	1	12	0	0	0	0	0	0	1	1	0	1	0	2	0	2	500	0	0	1
19	129919C	03/05/2002	0	17	1	24	1	12	1	12	0	0	0	0	0	0	0	0	1	1	0	0	0	2	0	2	1600	0	1	0
20	688903C	09/09/2005	0	17	1	1	1	2	1	1	0	0	1	1	0	0	0	0	1	1	0	0	0	1	0	1	500	0	0	0
21	099680D	18/09/2007	0	16	1	1	1	1	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	2	0	2	400	0	0	0
22	289722D	21/08/2008	0	14	1	6	1	6	0	0	0	0	1	3	0	0	0	0	1	1	1	1	0	3	1	4	600	0	0	0
23	546589C	22/11/2004	0	13	1	4	1	4	1	4	0	0	0	0	0	0	0	0	1	1	0	0	0	2	1	2	750	0	0	0
24	192403C	09/03/2002	0	11	1	3	1	10	1	6	0	0	0	0	0	0	0	0	1	1	1	1	0	3	0	2	250	0	0	0
25	030815D	07/11/2007	0	20	1	24	1	24	1	24	0	0	1	24	0	0	0	0	1	1	0	0	0	2	1	4	800	1	0	0
26	933376C	12/07/2006	1	13	1	1	1	1	0	0	1	1	1	1	0	0	0	0	1	1	0	0	0	3	1	8	1000	0	0	0
27	797561B	30/09/1999	1	19	1	3	1	3	1	3	1	6	0	0	0	0	0	0	1	1	0	0	0	1	0	3	600	1	0	0
28	992502C	16/05/2007	0	13	1	1	1	1	0	0	0	0	1	1	0	0	0	0	1	1	0	0	0	3	1	7	400	0	0	0
29	836850B	18/01/2000	1	18	1	12	1	12	1	12	0	0	0	0	0	0	0	0	1	1	0	0	0	2	0	5	800	0	1	0
30	702003C	11/03/2005	0	13	1	12	1	3	1	3	0	0	1	2	0	0	0	0	1	1	1	1	0	3	1	4	2000	0	0	0

31	772319C	03/10/2006	0	12	1	6	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	0	0	3	1	4	300	1	0	0
32	143727C	04/10/2002	0	16	0	0	1	10	1	10	1	6	1	10	0	0	0	0	1	1	0	0	0	1	0	1	1300	0	0	0
33	981898C	03/02/2007	0	22	0	0	1	12	1	12	1	12	1	12	0	0	0	0	1	1	0	0	0	2	0	5	900	1	1	0
34	869083C	09/05/2006	0	17	1	3	1	3	1	3	0	0	0	0	0	0	1	2	1	1	0	0	0	2	0	1	300	0	0	0
35	956232B	14/11/2000	0	15	1	2	1	2	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	2	0	2	1500	0	0	0
36	995342C	04/04/2007	0	15	1	12	1	12	1	12	0	0	0	0	0	0	0	0	1	1	1	0	0	4	1	7	3000	1	0	0
37	305811D	25/09/2008	0	17	1	18	1	12	0	0	0	0	1	12	0	0	1	2	1	1	1	1	0	4	1	8	2000	0	1	0
38	669603B	15/10/1998	0	14	1	3	1	2	1	2	0	0	1	2	0	0	0	0	1	1	0	0	0	2	0	2	500	1	0	0
39	482841C	13/07/2004	1	18	1	36	1	24	1	12	1	12	1	12	0	0	0	0	1	1	1	0	0	4	1	5	5000	1	1	0
42	946311C	01/11/2007	0	24	1	6	1	6	1	6	1	6	0	0	0	0	0	0	1	1	0	0	0	1	0	1	200	0	0	0
40	863844C	22/08/2006	0	13	1	1	1	1	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	2	1	1	500	0	0	0
41	833488C	13/07/2006	0	24	1	3	1	3	1	3	0	0	1	3	0	0	0	0	1	1	0	0	0	2	1	2	500	0	0	0
43	533029C	26/10/2004	0	23	1	12	1	12	1	12	0	0	0	0	0	0	0	0	1	1	0	1	0	3	1	8	2000	1	0	0
44	841751B	01/05/2000	0	36	1	12	1	12	1	12	0	0	0	0	0	0	0	0	1	1	0	0	0	2	1	4	400	0	0	0
45	845322C	07/03/2006	1	16	1	3	1	3	0	0	0	0	1	3	0	0	0	0	1	1	0	0	0	3	1	1	900	0	0	0
46	129501C	04/05/2002	1	10	1	3	1	3	1	2	1	3	0	0	0	0	0	0	1	1	1	1	0	4	1	5	2000	1	0	0
47	670186B	20/10/1998	0	22	1	12	1	12	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	2	0	2	300	0	0	0
48	000758C	27/03/2001	0	19	1	6	1	6	1	6	1	6	0	0	0	0	0	0	1	1	1	0	0	3	0	5	2200	1	0	0
49	296310D	16/09/2008	0	15	1	18	1	12	0	0	0	0	1	12	0	0	1	6	1	1	1	1	0	3	1	3	700	1	0	0
50	755910C	23/01/2006	0	14	1	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	1	1	1	700	0	0	0
51	245949C	15/01/2003	0	21	1	2	1	2	1	2	0	0	0	0	0	0	0	0	1	1	0	0	0	2	1	2	600	1	0	0
52	937451B	26/09/2000	0	20	1	1	0	0	0	0	1	1	0	0	0	0	0	0	1	1	0	0	0	3	0	2	1100	1	1	0
53	017660D	05/10/2007	0	18	1	3	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	3	1	6	800	0	0	0
54	900189B	30/06/2000	0	17	1	1	1	24	0	0	0	0	0	0	0	0	0	0	1	1	0	1	0	3	0	5	1800	1	1	0
55	155470C	16/05/2002	1	11	1	4	1	8	0	0	0	0	0	0	0	0	0	0	1	1	1	0	0	3	0	5	1800	0	0	0
56	307481C	14/08/2003	0	18	1	1	1	4	1	4	0	0	0	0	0	0	0	0	1	1	0	0	0	2	0	2	750	1	1	0
57	161274C	05/02/2006	0	16	1	5	1	5	1	5	0	0	0	0	0	0	0	0	1	1	0	0	0	2	1	1	800	0	0	0
58	280247D	08/07/2008	0	11	1	5	1	5	0	0	0	0	0	0	1	3	0	0	1	1	1	0	1	4	1	7	500	0	0	0
59	168407C	07/09/2002	1	17	1	6	1	6	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	1	0	2	300	0	0	0

NO	NAME	H.NO	NC	SPF	NPX	MS	ES	SS	TPF	PP	BASE	INTER	ITF	IOF	SOF	OA	OC	PPS	FO	FR	FL	EXTRA	CS	ID	PIT	ACF	CLI	CRIB
1	ABHIGIT ROY	031300D	1	0	1	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	ABHIJIT MANDAL	837122C	1	1	1	0	1	1	1	0	1	0	1	1	0	1	0	0	0	1	0	1	1	0	0	1	0	1
3	ABISHEK KUMAR	080434C	1	1	1	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
4	ARDHENDU HATI	852268C	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
5	BALASUBRAMANIAM.B.C.	257706C	1	1	1	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
6	BENIMADHAB	642674B	1	1	1	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
7	CHUTTAN RAJWAR	940359B	1	1	1	1	1	1	1	1	1	1	1	1	1	1	0	1	0	1	0	1	1	0	0	0	0	0
8	DEB KUMAR SANTRA	190856D	1	1	1	1	1	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
9	DHANANJAY KUMAR	283606D	1	1	1	0	0	1	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
10	DHARMENDRA	928021B	1	1	1	0	1	1	1	1	1	1	1	1	0	0	0	1	1	1	0	1	1	0	0	1	1	0
11	DIPANKAR MAJUMDAR	992544B	1	1	1	0	0	1	1	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
12	GOUTAM KUMAR MAITY	742758B	1	1	1	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
13	JATIN MAJI	617396C	1	1	1	1	1	1	1	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
14	JAYANTA KONAR	311077C	1	1	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
15	KAUSHAL KUMAR	945376C	1	1	1	1	1	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
16	KIRAN BAG DAS	985194B	1	1	1	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
17	KUMAR	639751c	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
18	MADHABANANDA	310594C	1	1	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
19	MAHADEB SAUTYA	129919C	1	1	1	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20	MANOJ DHUA	688903C	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
21	MD MOQARIM	099680D	1	1	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
22	MITHILESH RUPAL SHARMA	289722D	1	1	1	1	0	1	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
23	MOHAMMED SADDAM HUSSAIN	546589C	1	1	1	0	1	1	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
24	NARAYAN DAS	192403C	1	1	1	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
25	NAVIN KUMAR	030815D	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
26	PALLAB JOSH	933376C	1	1	1	0	1	1	1	0	0	1	1	1	0	1	0	1	1	0	0	1	0	0	0	0	0	0
27	PARANTHAMAN	797561B	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
28	PRABU R.	992502C	1	1	1	0	1	1	1	0	1	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
29	PRADEEP KU RATREY	836850B	1	1	1	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
30	PRAVEEN KUMAR PANDE	702003C	1	1	1	0	1	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
31	RAHUL KARMAKAR	772319C	1	0	1	1	0	1	0	0	1	0	1	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0
32	RAJKUMAR BEJ	143727C	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
33	RANJIT GHORAI	981898C	1	1	1	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
34	RAVI SHANKAR	869083C	1	1	1	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
35	REDDAPPA	956232B	1	1	1	0	1	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

36	ROHIT KUMAR	995342C	1	1	1	0	1	1	1	1	1	1	1	1	1	1	0	1	1	1	0	1	1	0	0	0	0	0
37	SABU CHANDRA ROY	305811D	1	1	1	0	1	1	1	0	1	0	1	1	1	1	0	0	0	1	0	1	1	0	0	0	0	0
38	SAMIR NARAYAN JAMADAR	669603B	1	1	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
39	SANJIB XESS	863844C	1	0	1	0	1	1	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
42	SANJIV KUMAR	482841C	1	1	1	0	1	1	1	1	0	0	1	0	1	1	0	1	0	1	0	1	1	0	0	0	0	0
40	SANJOY GHOSH	946311C	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
41	SATHEESH KUMAR	833488C	1	1	1	0	0	1	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
43	SENTHIL	533029C	1	1	1	0	1	1	1	0	1	0	1	1	0	1	0	0	0	1	0	0	0	0	0	0	0	0
44	SHANMUGAM	841751B	1	1	1	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
45	SHASHI SHAW	845322C	1	1	1	0	0	1	1	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
46	SHEIK AFSAR	129501C	1	1	1	0	0	1	1	0	0	0	1	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0
47	SHYMAL SAHA	670186B	1	1	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
48	SIVA KUMAR	000758C	1	1	1	0	0	1	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
49	SK. BILAL HOSSAN	296310D	1	1	1	0	1	1	1	0	1	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
50	SUDIP GHORAI	755910C	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
51	SUJOY SARKAR	245949C	1	1	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
52	SUMAN DAS	937451B	1	1	1	1	1	1	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
53	SUMAN PAL	017660D	1	1	1	0	1	1	1	0	1	0	1	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0
54	SUMED KUMAR MONDAL	900189B	1	1	1	0	1	1	1	0	0	1	1	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0
55	SUMIT DAS	155470C	1	1	1	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
56	SURESH M	307481C	1	1	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
57	TANMOY CHOWDHARY	161274C	1	1	1	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
58	UTTAM DAS	280247D	1	1	1	1	1	1	1	0	1	0	1	1	1	1	0	0	0	1	0	1	1	0	0	0	0	0
59	VENKETESAN	168407C	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

NO	NAME	HOSPITAL NO	DOS	EMB	I/L IMA	B/L IMA	APA	OPH	VIDI	MENIN	CAVER	PVA	GELF
1	ABHIGIT ROY	031300D	20/6/2007	1	0	1	0	0	0	0	0	0	1
2	ABHIJIT MANDAL	837122C	10/8/2006	1	1	0	0	0	0	0	0	1	1
3	ABISHEK KUMAR	080434C	23/10/2001	0	0	0	0	0	0	0	0	0	0
4	ARDHENDU HATI	852268C	11/7/2006	0	0	0	0	0	0	0	0	0	0
5	BALASUBRAMANIAM.B.C.	257706C	21/02/2003	0	0	0	0	0	0	0	0	0	0
6	BENIMADHAB	642674B	3/8/1998	0	0	0	0	0	0	0	0	0	0
7	CHUTTAN RAJWAR	940359B	10/1/2000	1	0	1	1	1	1	1	0	0	1
8	DEB KUMAR SANTRA	190856D	21/4/2008	0	0	0	0	0	0	0	0	0	0
9	DHANANJAY KUMAR	283606D	9/10/2008	1	0	1	0	0	0	0	0	0	1
10	DHARMENDRA	928021B	7/3/2002	1	0	1	1	0	0	0	0	0	1
11	DIPANKAR MAJUMDAR	992544B	9/3/2001	1	0	1	0	1	0	0	0	0	1
12	GOUTAM KUMAR MAITY	742758B	11/6/1999	1	1	0	1	0	1	0	0	0	1
13	JATIN MAJI	617396C	7/4/2005	1	0	1	0	0	0	0	0	0	1
14	JAYANTA KONAR	311077C	4/6/2003	0	0	0	0	0	0	0	0	0	0
15	KAUSHAL KUMAR	945376C	17/01/2007	1	0	1	0	0	0	0	0	0	1
16	KIRAN BAG DAS	985194B	2/2/2001	0	0	0	0	0	0	0	0	0	0
17	KUMAR	639751c	10/6/2005	0	0	0	0	0	0	0	0	0	0
18	MADHABANANDA	310594C	1/7/2003	0	0	0	0	0	0	0	0	0	0
19	MAHADEB SAUTYA	129919C	5/3/2002	0	0	0	0	0	0	0	0	0	0
20	MANOJ DHUA	688903C	9/9/2005	0	0	0	0	0	0	0	0	0	0
21	MD MOQARIM	099680D	18/9/07	0	0	0	0	0	0	0	0	0	0
22	MITHILESH RUPAL SHARMA	289722D	21/08/2008	1	0	1	0	0	0	0	0	0	1
23	MOHAMMED SADDAM HUSSAIN	546589C	22/11/2004	1	0	1	0	1	0	0	0	0	1
24	NARAYAN DAS	192403C	3/9/2002	0	0	0	0	0	0	0	0	0	0
25	NAVIN KUMAR	030815D	11/7/2007	1	0	1	0	0	0	0	0	0	1
26	PALLAB JOSH	933376C	7/12/2006	1	1	0	0	0	1	0	0	0	1
27	PARANTHAMAN	797561B	30/09/1999	0	0	0	0	0	0	0	0	0	0
28	PRABU R.	992502C	16/5/2007	1	0	1	0	0	0	0	0	0	1
29	PRADEEP KU RATREY	836850B	18/1/2000	0	0	0	0	0	0	0	0	0	0
30	PRAVEEN KUMAR PANDE	702003C	3/11/2005	1	0	1	1	0	0	0	0	0	1
31	RAHUL KARMAKAR	772319C	10/3/2006	1	1	0	0	0	0	1	0	0	1
32	RAJKUMAR BEJ	143727C	10/4/2002	0	0	0	0	0	0	0	0	0	0
33	RANJIT GHORAI	981898C	2/3/2007	0	0	0	0	0	0	0	0	0	0
34	RAVI SHANKAR	869083C	5/9/2006	0	0	0	0	0	0	0	0	0	0

35	REDDAPPA	956232B	14/11/2000	0	0	0	0	0	0	0	0	0	0
36	ROHIT KUMAR	995342C	4/4/2007	1	0	1	0	0	0	0	0	0	1
37	SABU CHANDRA ROY	305811D	25/09/2008	1	0	1	1	0	0	0	1	0	1
38	SAMIR NARAYAN JAMADAR	669603B	15/10/1998	0	0	0	0	0	0	0	0	0	0
39	SANJIV KUMAR	482841C	13/07/2004	1	0	1	1	1	0	0	1	0	1
42	SANJOY GHOSH	946311C	11/1/2007	0	0	0	0	0	0	0	0	0	0
40	SANKIB XESS	863844C	22/8/2006	1	1	0	0	0	0	0	0	0	1
41	SATHEESH KUMAR	833488C	13/07/2006	1	1	0	0	0	0	0	0	0	1
43	SENTHIL	533029C	26/10/2004	1	1	0	0	0	0	0	1	0	1
44	SHANMUGAM	841751B	5/1/2000	1	0	1	0	1	0	0	0	1	0
45	SHASHI SHAW	845322C	3/7/2006	1	0	1	1	0	0	0	0	0	1
46	SHEIK AFSAR	129501C	5/4/2002	1	0	1	0	1	0	0	0	0	1
47	SHYMAL SAHA	670186B	20/10/1998	0	0	0	0	0	0	0	0	0	0
48	SIVA KUMAR	000758C	27/3/2001	0	0	0	0	0	0	0	0	0	0
49	SK. BILAL HOSSAN	296310D	16/09/2008	1	0	1	0	0	0	0	0	0	1
50	SUDIP GHORAI	755910C	23/01/2006	1	1	0	0	0	0	0	0	0	1
51	SUJOY SARKAR	245949C	15/1/2003	1	1	0	0	0	1	0	0	0	1
52	SUMAN DAS	937451B	26/09/2000	0	0	0	0	0	0	0	0	0	0
53	SUMAN PAL	017660D	10/5/2007	1	0	1	0	0	0	0	0	0	1
54	SUMED KUMAR MONDAL	900189B	30/06/2000	0	0	0	0	0	0	0	0	0	0
55	SUMIT DAS	155470C	16/5/2002	0	0	0	0	0	0	0	0	0	0
56	SURESH M	307481C	14/8/2003	0	0	0	0	0	0	0	0	0	0
57	TANMOY CHOWDHARY	161274C	2/5/2006	1	0	1	0	0	0	0	0	0	1
58	UTTAM DAS	280247D	7/8/2008	1	0	1	0	1	0	0	0	1	1
59	VENKETESAN	168407C	9/7/2002	0	0	0	0	0	0	0	0	0	0

NO	HOSPITAL NO	sym free 1st sx	DOS	RECUR	age	blood loss	EPISTAXI	D OF EPI	NA OBS	D OF NO	N DISCH	D OF ND	HEADCE	D HEADC	MOUHT B	D OF MB	OTALGIA	D OF OTA	BLIND	D OF BL	HL	D OF HL
1	642674B	30	25/03/2003	0	27	500	1	24	1	24	1	24	0	0	0	0	0	0	0	0	1	12
2	064023D	1	23/08/2007	1	16	2000	1	8	1	8	1	8	1	3	1	3	0	0	0	0	1	6
3	444588B	24	31/08/1999	0	15	600	0	0	0	0	0	0	1	2	0	0	0	0	0	0	0	0
4	992544B	3	13/07/2001	0	13	500	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0
5	991920C	36	30/03/2007	0	18	400	1	24	1	24	0	0	0	0	0	0	0	0	0	0	0	0
6	742758B	48	18/02/2005	1	19	3000	1	24	1	24	0	0	0	0	0	0	0	0	0	0	0	0
7	744243C	12	02/09/2006	0	15	1400	1	12	1	12	1	12	0	0	0	0	0	0	0	0	1	12
8	201733d	6	04/07/2008	0	11	600	1	4	1	4	1	4	1	4	1	4	0	0	0	0	0	0
9	985194B	5	01/08/2002	0	19	1200	1	5	1	5	0	0	0	0	0	0	0	0	0	0	0	0
10	984359C	12	18/04/2007	0	26	1200	1	12	1	12	0	0	1	8	0	0	0	0	0	0	1	12
11	960744C	12	23/01/2007	0	17	2000	1	5	1	6	1	6	0	0	1	6	0	0	0	0	0	0
12	933376C	7	16/10/2008	0	15	1700	1	12	1	12	0	0	0	0	0	0	0	0	0	0	0	0
13	797561B	5	14/06/2001	0	21	600	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0
14	836850B	13	22/05/2001	0	19	200	1	3	1	3	0	0	0	0	0	0	0	0	0	0	0	0
15	944326B	16	20/10/2000	1	15	700	1	9	0	0	0	0	0	0	0	0	0	0	0	0	0	0
16	567217B	18	24/10/2000	0	20	700	1	16	1	16	0	0	0	0	0	0	0	0	0	0	0	0
17	657486C	6	14/07/2005	0	34	300	1	24	1	24	1	24	1	24	0	0	0	0	0	0	0	0
18	842277B	8	28/01/2000	0	11	2000	1	8	1	8	0	0	0	0	0	0	0	0	0	0	0	0
19	492077C	3	20/08/2004	1	19	1300	1	5	1	5	0	0	0	0	0	0	0	0	0	0	0	0
20	697573C	4	15/09/2005	0	18	400	1	12	1	12	1	12	0	0	0	0	0	0	0	0	0	0
21	845322C	6	28/02/2007	0	17	800	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0
22	129501C	13	07/04/2003	0	11	1000	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0
23	822149C	2	22/06/2006	0	15	700	1	6	1	6	0	0	0	0	1	6	0	0	0	0	0	0
24	995943C	3	04/05/2007	0	14	4600	1	1	1	1	1	1	1	2	1	1	0	0	0	0	0	0
25	553305C	3	12/06/2005	0	17	800	1	2	1	2	0	0	0	0	0	0	0	0	0	0	0	0
26	509205C	12	18/08/2004	0	11	1000	1	12	1	12	0	0	0	0	1	12	0	0	0	0	0	0
27	403604C	8	01/09/2004	1	18	900	1	12	1	12	1	10	0	0	0	0	0	0	1	12	0	0
28	821089B	6	14/01/2000	0	22	400	0	0	1	6	0	0	0	0	0	0	0	0	0	0	0	0
29	155470C	2	13/08/2002	0	11	500	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0
30	168407C	15	16/03/2004	0	18	850	0	0	1	6	1	6	0	0	1	6	0	0	0	0	0	0

NAA	NPX MAS	CHEEK	SOFT PA	PR	stage	embolization	approach	na. synachae	fistula	infection	SEPTAL PORFORAION	ORONASAL FISTULA
1	1	0	0	0	2		0 2	1	0	0	0	0
1	1	1	1	0	4		1 7	1	1	0	0	0
0	0	1	1	0	3		0 3	1	0	0	0	1
1	1	1	0	0	3		0 2	1	1	0	0	0
1	1	0	0	0	1		0 2	0	0	0	0	0
1	1	0	0	0	4		0 8	0	0	1	0	0
1	1	0	1	0	4		1 7	0	0	1	0	0
1	1	1	0	0	3		1 4	0	0	0	0	0
1	1	0	0	0	4		0 5	1	1	0	0	0
1	1	0	0	0	2		1 4	1	0	0	0	0
1	1	0	1	0	3		1 7	0	0	0	1	0
1	1	0	0	0	4		0 4	0	0	0	0	0
1	1	0	0	0	2		0 2	1	1	0	0	0
0	1	0	0	0	2		0 2	1	1	0	0	0
1	1	0	0	0	3		0 4	1	0	0	0	0
1	1	0	0	0	3		1 5	0	0	0	0	0
1	1	0	0	0	3		1 4	1	0	0	0	0
1	1	0	1	0	2		0 5	0	0	0	0	0
1	1	1	0	1	4		1 8	0	0	0	0	0
1	1	0	0	0	2		0 3	1	0	0	0	0
1	1	0	0	0	3		0 4	1	0	0	0	0
1	1	1	0	0	3		0 5	0	0	0	0	0
1	1	0	0	1	4		1 7	0	0	0	0	0
1	1	1	0	1	4		1 8	0	0	0	0	0
1	1	0	0	0	1		0 1	1	1	0	0	0
1	1	0	0	0	2		0 2	1	0	0	0	0
1	1	0	0	0	2		1 1	0	0	0	0	0
1	1	0	0	0	1		0 2	0	0	0	0	0
1	1	0	0	0	1		0 1	0	0	0	0	0
1	1	0	1	0	1		0 2	0	1	0	0	0

NO	HOSPITAL NO	NC	SPF	NPX	MS	ES	SS	TPF	PP	BAS	INT	INF	IOF	SOF	OA	PPS	FO	FR	EXT	CS
1	642674B	0	1	1	0	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0
2	064023D	1	1	1	0	1	1	1	1	1	1	1	1	1	1	1	0	1	1	1
3	444588B	0	0	0	0	0	0	1	1	1	0	1	1	0	0	0	0	0	0	0
4	992544B	1	1	1	0	1	0	1	0	1	0	1	1	0	0	0	0	0	0	0
5	991920C	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
6	742758B	1	1	1	0	0	1	1	1	1	1	1	1	1	0	1	1	1	1	1
7	744243C	1	1	1	0	1	1	1	1	1	1	0	0	0	0	1	0	1	1	1
8	201733d	1	1	1	0	0	0	1	0	1	0	1	0	0	0	0	0	0	0	0
9	985194B	1	1	1	0	1	1	1	1	1	0	1	1	0	0	0	0	1	1	1
10	984359C	1	1	1	0	1	1	1	0	0	1	0	0	0	0	0	0	0	0	0
11	960744C	1	1	1	0	1	1	1	1	1	1	1	0	0	0	1	0	0	0	0
12	933376C	1	1	1	1	0	1	1	0	0	0	1	1	0	0	0	0	0	1	1
13	797561B	1	1	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
14	836850B	1	1	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
15	944326B	1	1	1	0	0	1	0	0	1	1	1	0	0	0	1	0	0	0	0
16	567217B	1	1	1	0	1	1	1	0	0	0	1	0	0	0	0	0	0	0	0
17	657486C	1	1	1	0	1	1	1	0	1	0	1	0	0	0	0	0	0	0	0
18	842277B	1	1	1	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0
19	492077C	1	1	1	1	1	1	1	0	1	0	1	1	0	1	0	0	1	1	1
20	697573C	1	1	1	0	1	1	1	0	1	0	0	0	0	0	0	0	0	0	0
21	845322C	1	1	1	0	0	1	1	0	1	0	1	1	0	0	0	0	0	0	0
22	129501C	1	1	1	0	0	1	1	1	1	1	1	0	0	0	1	0	0	0	0
23	822149C	1	1	1	0	0	1	1	0	1	0	1	1	1	1	0	0	1	1	1
24	995943C	1	1	1	1	1	1	1	0	1	0	1	1	1	1	0	0	1	1	1
25	553305C	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
26	509205C	1	1	1	0	0	1	0	1	1	1	0	0	0	0	0	0	0	0	0
27	403604C	1	1	1	0	0	1	1	0	1	0	0	0	0	0	0	0	0	0	0
28	821089B	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
29	155470C	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

30	168407C	1	1	1	0		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
NO	NAME			HOSPITAL NO		DOS		embolization		I/L IMA		B/L IMA		APA		OPHTH		VIDIAN		MENINGEAL		cavernous		PVA		gelform	
1	BENIMADHEB			642674B		25/03/2003		0		0		0		0		0		0		0		0		0	
2	BHOLA SANKAR YADAV			064023D		23/08/2007		1		1		0		1		0		0		0		0		1	
3	BISWAJIT GHUGHU			444588B		31/08/1999		0		0		0		0		0		0		0		0		0	
4	DIPANKAR MAJUMDAR			992544B		13/07/2001		0		0		0		0		0		0		0		0		0	
5	DIPU KUNDU			991920C		30/03/2007		0		0		0		0		0		0		0		0		0	
6	GOUTAM KUMAR MAITY			742758B		18/02/2005		0		0		0		0		0		0		0		0		0	
7	HARUNAR RASHID			744243C		02/09/2006		1		0		1		1		0		0		0		0		1	
8	KANHA GOWSWAMI			201733d		04/07/2008		1		1		0		0		0		0		0		0		1	
9	KIRAN BAG DAS			985194B		01/08/2002		0		0		0		0		0		0		0		0		0	
10	MOGIDUL ISLAM MOOLLA			984359C		18/04/2007		1		0		1		0		0		0		0		0		1	
11	MONORANJAN BISWAS			960744C		23/01/2007		1		0		1		0		0		0		0		0		1	
12	PALLAB JOSH			933376C		16/10/2008		0		0		0		0		0		0		0		0		0	
13	PARANTHAMAN			797561B		14/06/2001		0		0		0		0		0		0		0		0		0	
14	PRADEEP KU RATREY			836850B		22/05/2001		0		0		0		0		0		0		0		0		0	
15	PRAOSENJIT DEY			944326B		20/10/2000		0		0		0		0		0		0		0		0		0	
16	PRASAD			567217B		24/10/2000		1		0		1		0		0		0		0		0		1	
17	RAMESHWAR RAM			657486C		14/07/2005		1		0		1		0		0		0		0		0		1	
18	SANTOSH DEY			842277B		28/01/2000		0		0		0		0		0		0		0		0		0	
19	SANTOSH PANDIT			492077C		20/08/2004		1		1		0		0		0		0		1		0		1	
20	SANTU NONGRUM			697573C		15/09/2005		0		0		0		0		0		0		0		0		0	
21	SHASHI SHAW			845322C		28/02/2007		0		0		0		0		0		0		0		0		0	
22	SHEIK AFSAR			129501C		07/04/2003		0		0		0		0		0		0		0		0		0	
23	SIMON BABU			822149C		22/06/2006		1		1		0		0		0		0		0		0		1	
24	SK ASMOT ALI			995943C		04/05/2007		1		0		1		0		0		0		0		0		1	
25	SOURAV MODAK			553305C		12/06/2005		0		0		0		0		0		0		0		0		0	
26	SUDIPTA MUKHERJEE			509205C		18/08/2004		0		0		0		0		0		0		0		0		0	
27	SUDUN KU SHIL			403604C		01/09/2004		1		0		1		0		0		0		0		0		1	
28	SUJOY KUNDU			821089B		14/01/2000		0		0		0		0		0		0		0		0		0	
29	SUMIT DAS			155470C		13/08/2002		0		0		0		0		0		0		0		0		0	
30	VENKETESAN			168407C		16/03/2004		0		0		0		0		0		0		0		0		0	

CLINICAL SIGNS OF JNA

Fig.1. Mass in nasal cavity

Fig.2. Cheek swelling

Fig.3. Soft palate bulge

STAGE I TUMOUR

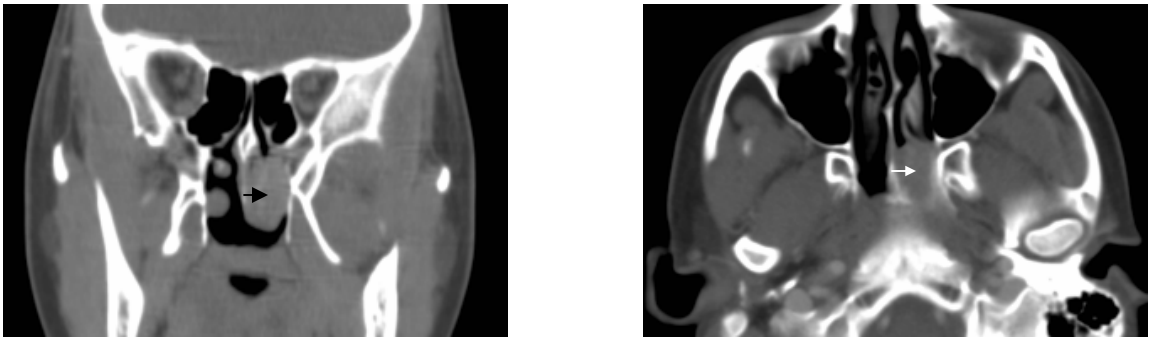

Fig.4a. Coronal CT scan shows a mass filling left nasal cavity (*black arrow*).**b.** Axial CT scan filling left nasopharynx (white *arrow).*

STAGE II TUMOUR

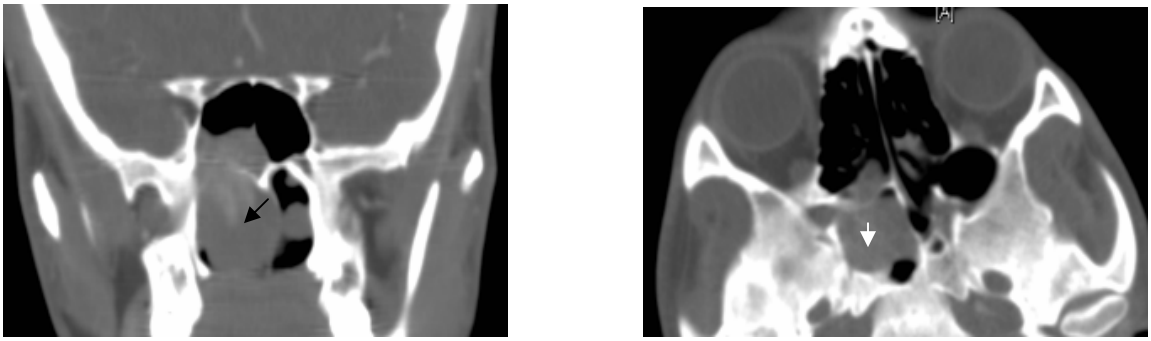

Fig.5a. Coronal CT scan shows a mass filling right nasal cavity and sphenoid sinus (*black arrow*).**b.** Axial CT scan filling right sphenoid sinus(white *arrow).*

STAGE III TUMOUR

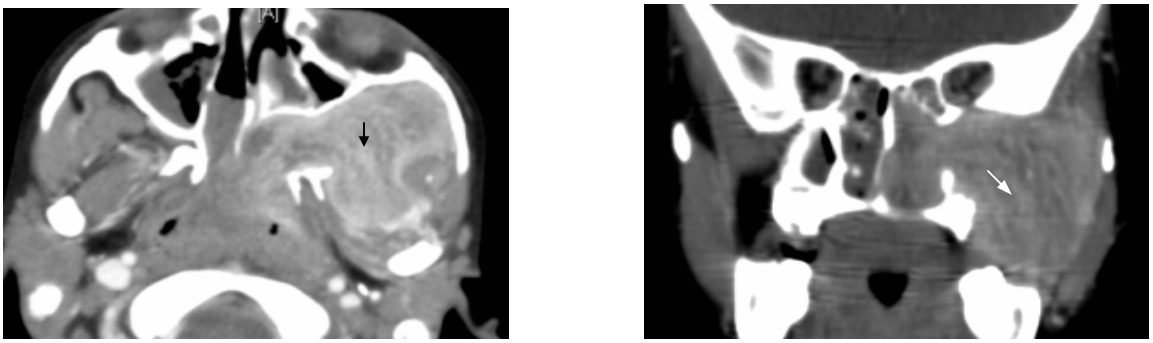

Fig.6a. Coronal CT scan shows a mass filling left nasal cavity, nasopharynx, pterygopalatine fossa and infratemporal fossa (*black arrow*).**b.** Axial CT scan filling left infratemporal fossa(white *arrow).*

STAGE IV TUMOUR

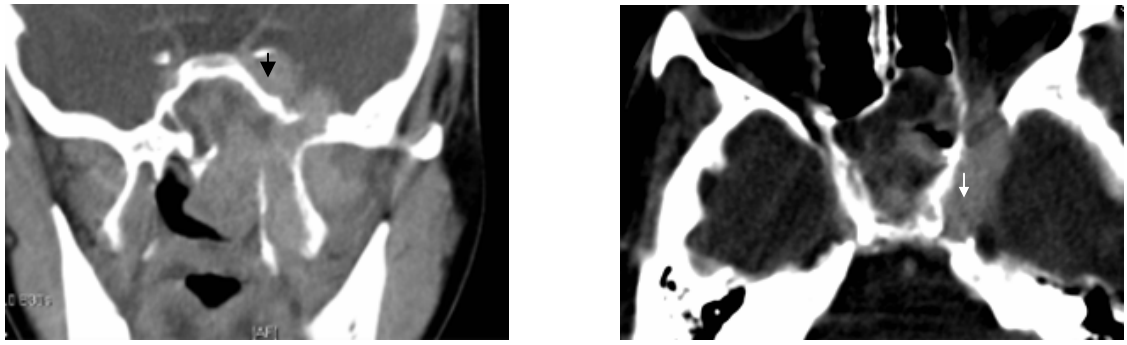

Fig.7a. Coronal CT scan shows a mass filling left nasopharynx, sphenoid sinus, right cavernous sinus(*black arrow*).**b.** Axial CT scan filling left cavernous sinus(white *arrow).*

ANGIOGRAM

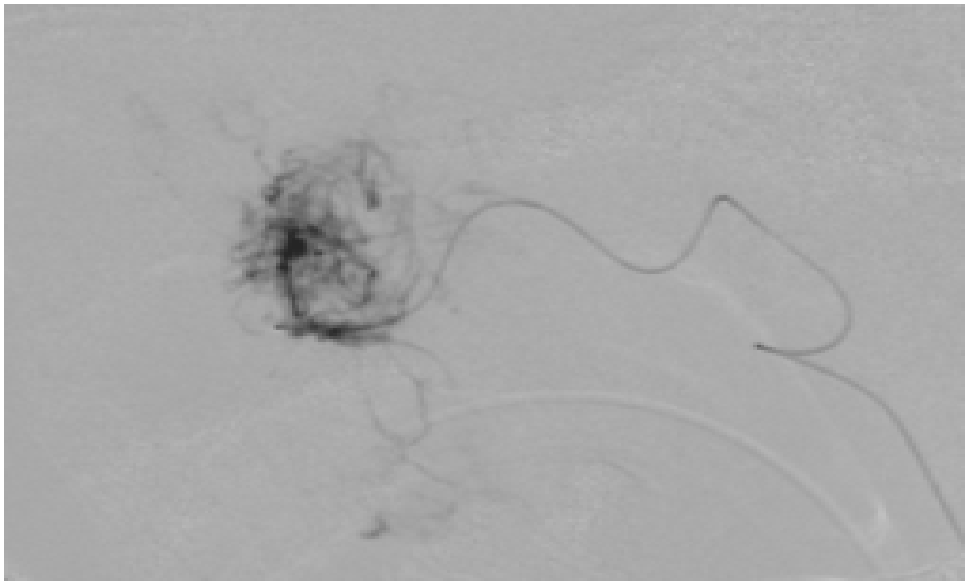

Fig.8. Angiogram shows abnormal tumor blush in the region supplied by left internal maxillary artery.

POST EMBOLIZATION ANGIOGRAM

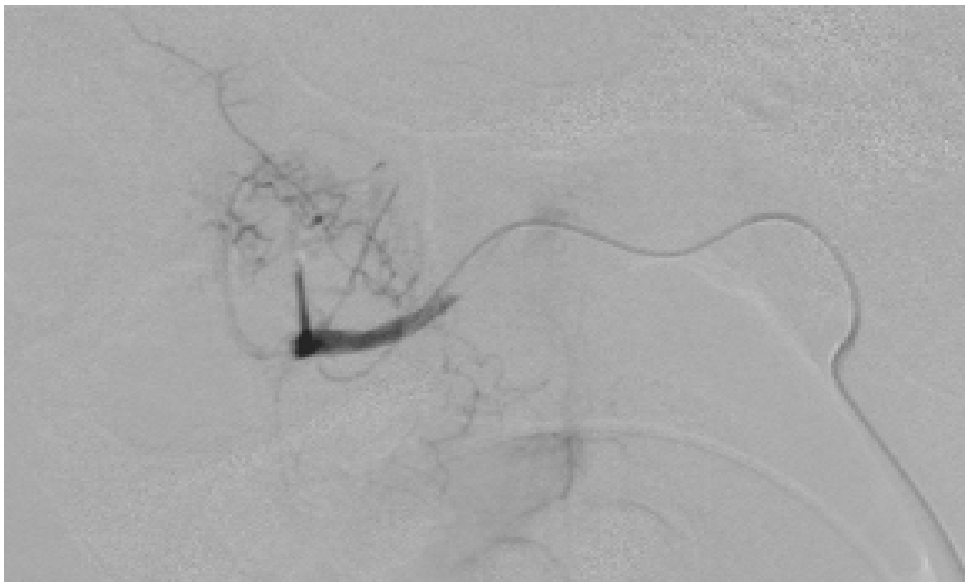

Fig.9. Post embolization check angiogram showed good occlusion of embolized vessel and absence of tumor blush.

SURGICAL APPROACHES

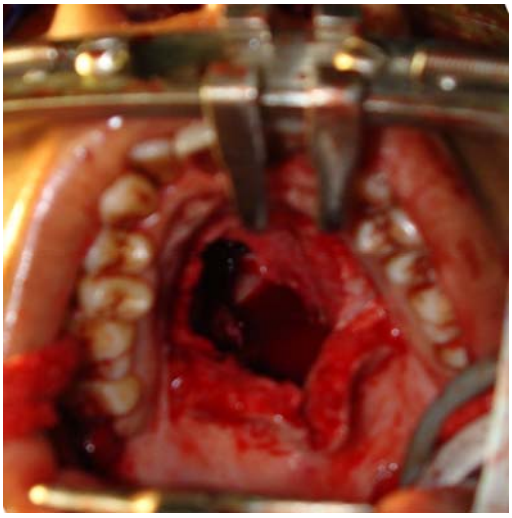

Fig.10. Transpalatal approach

Fig.11. Midfacial Degloving approach

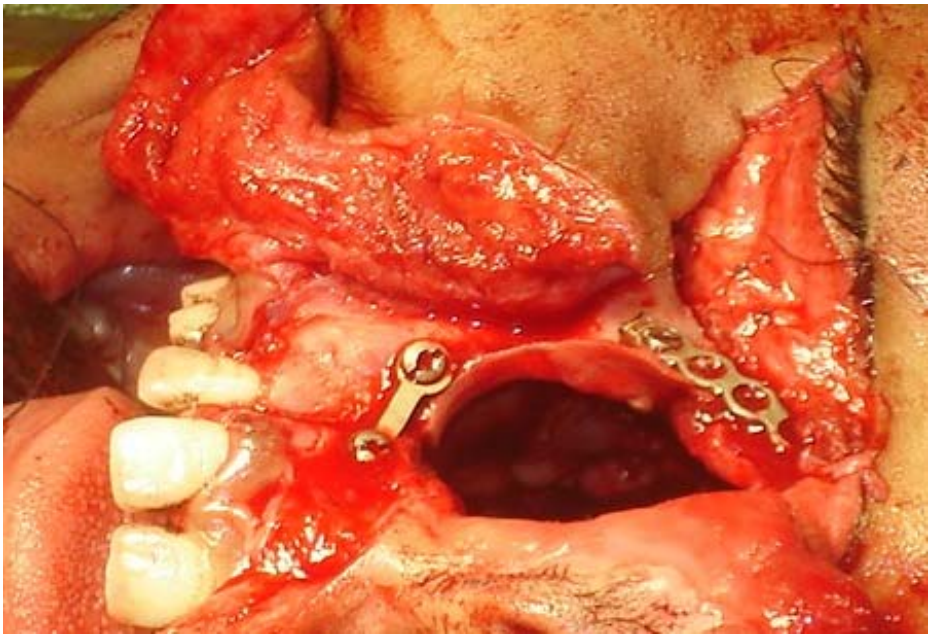

Fig.12. Extended osteoplastic maxillotomy approach

Fig.13. Lateral rhinotomy approach

Fig.14. Infratemporal fossa approach

SURGICAL SPECIMEN OF JNA

Fig.15. Typical pink, smooth surfaced, lobulated mass

POST OPERATIVE

Fig.16. Post operative lateral rhinotomy: Acceptable Cosmesis

Fig.17. Palatal fistula